Lumleian Lectures.

ON

CONVULSIVE SEIZURES.

Delivered before the Royal College of Physicians of London.

BY

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ON CONVULSIVE SEIZURES.

LECTURE I.

Definition of Terms.—Classification of Convulsions.—The Three Evolutionary Levels in the Nervous System.—Middle Level Fits. "Epileptiform."—Highest Level Fits: "Genuine Epilepsy."—Lower Level Fits: "Ponto-bulbar."—Varieties of Lowest Level Fits.—Abnormal Affections of Consciousness in different kinds of Fits.—The Discharging Lesion in Epileptic and Epileptiform Fits.—The Interconnecting Fibres.—The Kinetic Route.—Representation of Movements.—The Right Motor Region.—The Cerebellar System.

THERE are two pleasant preliminaries, the sole parts of my address I can enter upon with a light heart. The first is most earnestly to thank you, Mr. President, for the honour you conferred on me in asking me to deliver the Lumleian Lectures. There is one thing for which I cannot blame myself. I have, as was my duty, taken all the pains I could over the task you were so good as to assign to me. The second preliminary is to mention my great obligations to Dr. Ferrier. There are very few men of the day by whom both the art and science of medicine have been so greatly helped. It is only since his remarkable researches that medical men in this country have studied convulsions in a thoroughly realistic way. Without further particular acknowledgments I make the general one, that all over the field of neurology I am profoundly indebted to Ferrier.

Convulsions and other paroxysms are owing to (1) sudden, (2) excessive, and (3) temporary nervous discharges.[1] The term "nervous discharge" (used before me by Spencer) has been much objected to; when I say that it is used synonymously with "liberation of energy by nervous elements," it will mislead no one. There are nervous discharges in all the operations of health. I should use the term "explosive discharge" for the abnormal liberation of energy in convulsions were it not that physiologists speak of "explosive decomposition of the muscle's substance" which gives rise to contraction of the muscle in health; and thus it may be that normal nervous discharges, those in the operations of healthy people, are "explosive." So then I use the word "excessive" for the discharges which have the three characters mentioned, the words "sudden" and "temporary," or the word "paroxysmal," being understood; when dealing with convulsions, it will not be always necessary to add the term "excessive." I

[1] A term introduced by Dr. Edward Liveing in his masterly work *On Megrim* is "nerve storm." This term has met with much favour, and the conception it stands for has been of great value in elucidating some very complex problems in neurology.

shall, however, occasionally use "explosive" for states of cells of a discharging lesion, qualifying it by the word "highly," and sometimes the term "high instability." I do not speak of "disorderly discharges," for if in some ways the expression be correct, it hides from us the fact that the most brutal-looking convulsion is only the sign of a departure by a vast excess and by a caricature from normal nervous discharges.

I have always assumed, and shall continue to suppose, that convulsion results from excessive discharges of nerve cells, meaning, of course, liberation of energy during rapid decomposition (katabolism) of some matter in, or of part of, those cells. I shall frequently speak of cells concerned with excessive (primary) discharges as constituting a "discharging lesion," and sometimes of them as making up a "physiological fulminate," or occasionally, using Horsley's term, of their being together an "epileptogenous focus." Some material of the cells which make up the discharging lesion has, by morbid nutrition, become of very high tension and of most unstable equilibrium (briefly of high instability), and occasionally discharges excessively. I do not assert that excessive (primary) discharges producing convulsions always depend on a persistent state of high instability of cells; to give but one example to the contrary, excessive discharges beginning in a healthy rabbit's respiratory centres are induced by rapidly bleeding the animal to death, or by quickly asphyxiating it.

The discharging lesion, though I speak of it as persistent, meaning that it is of the same locality throughout each case, yet varies in its condition.[2] The discharges are occasional. After their excessive discharge the cells are no doubt far below the degree of stability (properly comparatively slight instability) of normal cells; they will reattain a highly abnormal degree of instability —again become highly explosive—by further morbid nutrition. These qualifying remarks should be borne in mind, and especially when the discharging lesion is spoken of as a fulminate—it is occasionally fulminant.

I make three classes or kinds of convulsions. Convulsions, or I will say, fits, differ in kind, according as centres discharged differ in rank; or, speaking more definitely, as the centres first engaged in paroxysms make up different evolutionary levels of the central nervous system. I have several times suggested that there are three levels of the cerebral (central) nervous system; each is sensori-motor, and each represents impressions and movements of all parts of the body. I speak briefly of what I suppose to be the hierarchy of centres of the nervous system as a basis for the classification of fits.

(1) The lowest or first level is roughly and incompletely defined as consisting of cord, medulla, and pons,[3] and more completely, and yet still roughly, as being that sensori-motor division of the central nervous system, to and from which pass nerves (all cranial and all spinal nerves) for every part of the body. This level, speaking of its motor elements, represents simplest movements of all parts of the body by a series of lowest motor centres (lowest motor centre being a proper name for a centre of the lowest level) from those in the aqueduct of Sylvius for simplest movements of the

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[2] The rapid reader must not take "persistent discharging lesion" to be "persistently discharging lesion."

[3] I have spoken briefly on what I believe to be the importance of reckoning the cord, medulla, and pons, or, rather, certain elements of these morphological divisions, as one, the lowest level—I believe it is the "spinal system" of Marshall Hall—(JOURNAL, July 14th, 1888). I do not pretend to be able to define the upper limit of this level.

ocular muscles to those of the sacral cord for simplest movements of the muscles of the perineum. This universally representing level is cerebro-cerebellar; it is at once the lowest level of the cerebral system and of the cerebellar system. For the present I ignore the higher levels of the cerebellar system, and go on to speak of two higher levels of the cerebral system. These levels are, as the lowest level is, sensori-motor, but I find it possible to illustrate by motor centres only, not, however, believing that these so-called "motor centres" are purely motor. (2) The middle or second level (its motor province) of the cerebral system is composed of centres of the Rolandic region (so-called "motor region" of the cerebral cortex), and, possibly, of the ganglia of the corpus striatum also. It represents complex movements of all parts of the body from eyes to perineum (re-represents). (3). The highest or third level (its motor province) of the cerebral system is made up of centres of the præfrontal lobes (highest motor centres, motor division of the "organ of mind"). It represents most complex movements of all parts of the body from eyes to perineum (re-re-represents). The highest centres (sensory and motor divisions of the highest level)—the "organ of mind," or anatomical substrata of consciousness—are the acme of the evolution; they have the same kind of constitution as lower centres; they are sensori-motor as certainly as the lumbar enlargement is. (Of course each level is bilateral).

That the lowest level is a very distinct division of the central nervous system will, I think, be granted. The separation of the frontal lobe into middle motor centres ("motor region"), making up the motor province of the middle level, and highest motor centres (præfrontal lobe) making up the motor province of the highest level, is, of course, hypothetical; there is no obvious morphological separation. I do not suppose that the evolutionary distinction is so abrupt or so decided as that between the middle and lowest levels. I will mention some differences in the two regions of the frontal lobes. (*a*) It seems certain that the middle motor centres ("motor region") are those cerebral centres directly connected with the lowest motor centres, as the facts of "Wallerian wasting" show; there is, however, possibly some "wasting" of fibres from the præfrontal lobes as low as the pons. This wasting has been differently interpreted. (*b*) The middle motor centres contain most large cells. (*c*) The middle motor centres are experimentally "excitable," and the præfrontal lobes are not. This is a very important difference; it may be taken to mean that the præfrontal lobes are not, as I suppose, motor. The distinction currently made is vastly greater than the one I have submitted. Believing that the whole central nervous system (the organ of mind included—the mind, of course, not included) is a sensori-motor mechanism of three levels, the distinction I make is not of kind but merely of degree—it is that the præfrontal lobe is only greatly more complex, etc., than the "motor region.' But, according to the received doctrine, whilst the latter alone is motor, the former differs from it in two ways; the præfrontal lobe has no motor constitution, and is part of the "intellectual centres." Distinguishing the psychical from the physical, I would say that psychical states are not functions of any centre, but are simply concomitant with functioning of the most complex, etc., sensori-motor nervous arrangements—those of the highest level ("organ of mind") of which level the præfrontal lobe is the motor division.

There are, I submit, three kinds of fits corresponding to the three evolutionary levels. It is convenient to speak of the three

kinds in an order different from that used in stating the levels. (2) I mention epileptiform seizures first because their localisation is not doubtful. (They were first described by Bravais in 1824.) They are "middle level fits"—that is, they are produced by excessive discharges beginning in parts of the middle level (motor province) of the cerebral system ("motor region"). My hypothesis is that (3) fits of epilepsy proper ("genuine epilepsy" of some nosologists) are "highest level fits," and that many of them, not all, are produced by excessive discharges beginning in parts of the præfrontal lobes, highest level (motor province) of the cerebral system. Although the præfrontal lobes are not experimentally excitable, I suppose it will not be denied that their cells katabolise and liberate energy in their normal activities, and it is not unreasonable to suppose that cells of parts of them may, by pathological changes, become highly explosive, so that they occasionally discharge excessively. So that, if the præfrontal lobes are divisions of the "intellectual centres," as no doubt they are, and not, as I think, motor too, excessive discharge beginning in parts of them may produce epileptic fits.

Of course, this is speculative. I am not aware that anyone pretends to know the seat or the pathology of cases of "genuine epilepsy." I do not use the term "cortical epilepsy," because both epilep*tic* and epilep*tiform* seizures are, to my thinking, cortical fits. (The difference in meaning assigned to the two terms epileptic and epileptiform must never be lost sight of.) (1) I think that there are "lowest level fits." These are fits produced by excessive discharges beginning in parts of the lowest level, a level which is common to the cerebral and the cerebellar systems. I suppose that most of them are owing to excessive discharges beginning in centres of the bulbar and pontal regions of the level, hence I sometimes use the term "ponto-bulbar fits." With regard to epileptiform and epileptic fits, I deal almost exclusively with cases of patients subject to fits—with, so to speak, "chronic cases."

I have so far spoken only of three Kinds of fits. No doubt there are Varieties of each kind. Whilst the kind answers to the level, the variety of each kind answers to the particular part of the level in which the excessive discharge begins. It is certain that there are varieties of (2) epileptiform seizures; each is marked by a particular place of onset of the convulsion. There must be at least as many varieties of (3) epilepsy proper as there are different "warnings" of the paroxysms. Presumably there are varieties of (1) ponto-bulbar fits.

Then, of course, there are Degrees of each variety dependent directly on degree of the primary discharge, and indirectly on that of the secondary discharges. (2) For example, there are degrees of that variety of epileptiform seizures marked by the first spasm being of the thumb. There are many ranges of it, from convulsion almost limited to a thumb, thence onward to universal convulsion. (3) Everybody recognises that epileptic attacks occur in two vastly different degrees, *les petits maux* and *les grand maux*, and there are sub-degrees of each of these degrees. (1) There are degrees of convulsion in lowest level fits produced by Brown-Séquard's method in guinea-pigs. There are, no doubt, degrees of respiratory fits produced in certain lower animals by rapid bleeding, by ligature of the great arteries of the neck, and by sudden stoppage of respiration.

To repeat, "fit" is a term used to include convulsive paroxysms of all kinds dependent on excessive discharges beginning in any part of any one of the three levels, the epileptic and epileptiform

beginning in one half [4] of a level. There are three kinds of fits, (1) ponto-bulbar, (2) epileptiform, and (3) epileptic. There are, the supposition is, varieties of each kind, and degrees of each variety of each kind.

I have used the wide term "fit" advisedly because my method is not merely an empirical or clinical one. It is not only an endeavour to find out whether a convulsive paroxysm a patient has is like or unlike that of the type "genuine epilepsy" of nosologists or any other type, but is also an endeavour to discover how it shows a particular departure from normal states of his nervous system.

Is there any difficulty in recognising the difference in the two mental attitudes, or, as I shall now say, the distinctness of the two view-points? From the empirical or clinical view-point we look to see how this or that fit approaches this or that nosological type. From the scientific view-point we look to see how these or those paroxysmal manifestations are produced, asking ourselves, "What is the level and what is the particular part of it in which the excessive discharge producing this or that set of manifestations begins?" or, regarding all kinds of fits, the more general question, "What different effects can an excessive discharge 'get out of' the different levels?"[5] The use of the general term "fit," compels careful segregation of kinds and analysis of individual cases. In this frame of mind we note the manifestations whether they have "the characters of an ordinary epileptic fit" or not. Whilst for purely scientific purposes I care very little for an answer to the question, "Do excessive discharges beginning in ponto-bulbar centres produce paroxysms resembling those of the epilepsy of nosologists?" I care very much for one to the question, "What effects do excessive discharges beginning in ponto-bulbar centres produce?" This question is, as yet, only to be replied to by experimenters who artificially produce fits in lower animals. I should be very much astonished if it turns out that excessive discharges beginning in any centres of the lowest level do produce convulsions having the same characters as those produced by such discharges beginning in centres of the higher levels. It would be marvellous if excessive discharges beginning in centres lowest in rank produced fits like those (epileptiform seizures) which are produced by excessive discharges beginning in parts of the more evolved centres, the middle motor cerebral centres ("motor region")[6].

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[4] It is convenient to use the word "half" (lateral) for nervous system and "side" for body.

[5] I formerly used the term epilepsy generically for all excessive discharges of the cortex and their consequences. At that time I did not think there were any fits in depending on excessive discharges beginning in any part of the ponto-bulbar centres. Using then the term epilepsy generically, I submitted that any part of the cerebral cortex might become highly over-unstable and discharge excessively. So that under the term epilepsy used generically there were epilepsy proper, epileptiform seizures, and migraine (the last mentioned being then spoken of as a sensory epilepsy), and, indeed, any paroxysmal symptoms attributable to sudden excessive discharges of any part of the cortex. I now use the term epilepsy for that neurosis, which is often called "genuine" or "ordinary" epilepsy, and for that only. Of course in all quotations I preserve the term epilepsy when used regarding any class of fits.

[6] Long ago (*St. And. Med. Grad Trans.*, vol. iii, 1870) I had the same mental attitude. After speaking of observations of the local onset and march of spasm in cases of fits from cerebral tumour, at a time when the cortical "motor region" had not been defined, I wrote: "We do not care to say that a tumour of the brain (or minute changes near it) had 'caused epilepsy,' but that changes in a particular region of the nervous system—say in the region of the middle cerebral artery—led to convulsions in which the spasm began in the right hand, spread to the arm, attacked next the face, then the leg, etc."

As I shall not have time to deal with lowest level fits, I will here mention what I think are some fits of this kind, not classifying them, but making a rough arrangement into three groups: 1. *Respiratory fits* (respiratorily beginning from primary discharge of the main (medulla) respiratory centre.[7] I think that fits of laryngismus stridulus come in this category, but Semon thinks they are cortical seizures.[8] Respiratory fits are easily induced in animals, and are described in all works on physiology when asphyxia is considered. Kussmaul-Tenner fits are respiratory fits.[9] It is said that convulsions occur in newborn animals after division of the cord below the medulla when they are asphyxiated; if so, these are certainly lowest level fits if not respiratory; it is possible that they are owing to discharge of subordinate (spinal) respiratory centres. 2. *Fits produced by convulsant poisons* (fits from nitrous oxide and curara are respiratory fits). Fits are experimentally produced in animals by absinthe and camphor. According to Magnan, convulsions are produced in animals by absinthe when the cerebrum has been removed. Many years ago Dr. George Johnson showed that convulsions occur in man from poisoning by camphor. Possibly some fits in renal disease (some so-called uræmic fits), and, it may be, fits occasionally part of a constitutional disturbance after urethral lesions, are ponto-bulbar fits from home-made poisons, as those just mentioned are supposed to be from foreign poisons. 3. *A condition for fits consequent on certain injuries of the cord or sciatic nerve* in guinea-pigs (Brown-Séquard). These are so well known that mere mention will suffice. There are often fits attending the onset of infantile paralysis; these are very difficult of explanation. I submit the hypothesis that they are lowest level fits produced by action on the ponto-bulbar centres of ptomaines, the result of disintegration of nervous matter of anterior horns; if so, they come in Group 1.

In all severe lowest level fits it is supposed that the primary discharge of ponto-bulbar centres not only induces discharge of other lowest motor centres, but also that by intermediation of sensory ("ascending") fibres it discharges centres of higher levels. (I never thought of implication of higher centres in these or any other fits by intermediation of sensory nerves until after consideration of the researches of Victor Horsley and Binswanger).[10]

It is necessary now to speak of abnormal affections of consciousness with regard to kinds of fits. I presume that there is loss of it in severe fits of all kinds. Consciousness is not a function of the highest cerebral centres; it is simply concomitant

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[7] *Brain*, April, 1886.

[8] See an able paper by Dr. Gay in *Brain*, January, 1890, for much valuable information on laryngismus stridulus, and for arguments against the view I take of the causation of the paroxysms.

[9] The fits produced in lower animals by rapid bleeding, by ligaturing the great arteries of the neck, and by asphyxia, are alike respiratory fits; they all depend on stimulation of the respiratory centres by lack of oxygen. Asphyxia experimentally produced in animals does not produce cerebral convulsions, but, on the contrary, renders the cortex inexcitable, whilst at the same time it increases the excitability of the ponto-bulbar centres. Again, as Franck says (*Fonctions Motrices du Cerveau*, pp. 86, 87), "l'anémie soit totale, soit partielle de l'encéphale, n'est nullement la cause des convulsions épileptiformes." Arrest of the heart by excitation of the vagus stops these fits.

[10] Horsley (*Lancet*, December 25th, 1886, abstract of Brown Lectures) believes that "all the convulsive, tonic, and clonic phenomena may originate from the ordinary bulbo-spinal centres such as exist for carrying out normal mechanisms. Tonic or clonic spasm, then, may be produced by any motor centre, but the combination and sequence of tonic-clonic could originate only from the cerebral motor cortex." This is important with regard to the question of ponto-bulbar fits.

with their functioning. There is no physiology of the mind any more than there is psychology of the nervous system. On the basis of mere concomitance, mental symptoms (synonymously abnormal states of consciousness) are, strictly speaking, only signs to physicians of what is not going on or of what is going on wrongly in part of a patient's material organisation. Thus cessation of consciousness at, or close upon, the onset of an epileptic fit is of value to physicians as a sign that the correlative physical process, the excessive discharge, begins in some part of the "organ of mind," or equivalently, highest centres of the cerebral system; the physical process in these and all other kinds of fits is our proper concern as medical men. Consciousness is lost late in epileptiform seizures, and in those of but little range there may not be even defect of consciousness; this agrees with the empirical evidence that the excessive discharge begins in lower (middle motor) centres; probably excessive discharges are induced (upwards) in the highest sensori-motor centres by intermediation of sensory fibres when consciousness begins to cease in an epileptiform seizure. Availing ourselves of abnormal affections of consciousness as signs of states of the central nervous system, we next, so to speak, put them on one side in order to study the process in fits in a purely materialistic manner.

We must bear in mind that not only is consciousness absent in negative functional states of the highest centres, but also that it ceases during the diametrically opposite functional state, excessive discharge beginning in those centres; there is loss of consciousness not only during, but also for some time after, a severe epileptic fit; in post-epileptic states there is temporary exhaustion of elements of the highest centres, and, corresponding to that exhaustion, there is absence of consciousness. There are, however, degrees of negative affection of consciousness. There are degrees from that slight defect in some fits of epilepsy (*les petits maux*) to seemingly entire loss of it in severe epileptic paroxysms; and there are degrees from that existing with trivial confusion of thought after a very slight epileptic fit to seemingly loss of all consciousness in deep coma after a very severe one. I now return to the physical process of fits.

It will be observed that I have spoken of the excessive discharge productive of fits beginning in this or that level; further, of its beginning in some part of a level. In recapitulation, the primary discharge in all kinds of fits is of some part of but one of the levels. And now I add that in epileptic and epileptiform seizures, of which alone I speak in the remainder of this lecture, the excessive discharge begins in some part of one half (lateral) of a level; thus, so to say, in these two kinds of fits the discharging lesion is "doubly local." If the discharging lesion be, as I suppose, of but a few cells, very little of a convulsion is directly due to it. Most of the convulsion is produced by intermediation of fibres between the cells of the discharging lesion and other cells of its own level and of other levels; there are induced, consecutive discharges of normal stable cells. Hence the interconnecting fibres of each level and the fibres connecting the several levels with one another, and the fibres connecting the lowest level with all parts of the body (lowest level of the whole organism), have to be considered. (I am straining the meaning of the word fibre, making it stand for any kind of nervous pathway ensuring physiological union, definite or indefinite.) Consideration of these connections is essential for clear ideas of the full process in fits, the only visible part of which is convulsion; it is especially important with regard to the way by which in epileptiform seizures a very local (a "doubly local") dis-

charging lesion causes wide-spreading and even universalisation of convulsion. Again, the study of the interconnections of the levels is a necessary preliminary to the comparison and contrast of the effects of "discharging lesions" and of "destructive lesions," an essential thing in the scientific investigation of diseases of the nervous system, as I urged in my Gulstonian Lectures (1869). I speak only of connections of motor centres of the levels.

Each level being bilateral is a twin series (right and left) of centres. There are connections (commissures) between "identical" centres and between "non-identical" centres of its two halves—presumably between centres of the two halves as they correspond for co-operation of the parts of the body they represent in joint operations by the two sides of the body. There are also connections between the centres making up the lateral half of each level. The fibres of the two connections spoken of are Intrinsic fibres of levels. I speak next of Extrinsic fibres—that is, of those interconnecting levels. Considering for a moment all the levels, the motor path,[11] or, as I shall say, kinetic route, extends from the highest motor centres to the muscles, which in a certain regard, being dischargeable, are centres too. This route (strictly the three series of motor centres are parts of the kinetic route) is in three segments: from highest to middle centres, first segment; from middle to lowest, second segment; and from lowest to muscles, third segment. I can, however, consider in detail only the second segment of the kinetic route, that connecting the middle and lowest levels. I shall, for convenience, speak of the motor centres of the right half of the middle level. There are three sets of motor fibres, kinetic lines of the second segment, uniting all right middle motor centres to all lowest motor centres —at least to all motor centres of the left half of the lowest level, if not, as I imagine, to those of the right also.

First Set of Fibres of the Second Segment.—Those which have been traced (on the Wallerian method by Charcot and others) from the right "motor region" along the right corona radiata, right internal capsule, through the right crus cerebri, right halves of pons and medulla into the (left) lateral column of the cord as low, Sherrington has found in one case, as the origin of the coccygeal nerve roots. These fibres are (and so are the second and third sets) extrinsic of the levels; they belong to neither level, and yet they belong to both in the sense of interconnecting the two.

Second Set of Fibres.—Those of the direct pyramidal tract. They have been said to be traceable no lower than the mid-dorsal region. Tooth has traced them by the Wallerian method in one case as low at least as the second lumbar; these are fibres of the inner part of the right anterior (Turck's) column.

Third Set of Fibres, comparatively recently (1884) discovered by Pitres, and seen by Schäfer, Sherrington, Hadden, Tooth, France, and others.[12] These fibres have been traced, on the Wallerian

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[11] Dr. Gowers (*Dis. of Nervous System*, vol. i, p. 116) gives a diagram of the "motor path." He makes two segments, "cerebro-spinal" and "spino-muscular." The kinetic route is a modification of his scheme.

[12] Mr. E. P. France (*Phil. Trans.*, B. 48, 1889) has not found after lesions of the marginal convolution in monkeys (made in some very important researches by Schäfer, Horsley, and Sanger-Brown, *Phil. Trans.*, B., 1888), nor in any other case in these animals degeneration of the direct pyramidal tract; but in all cases in which the degeneration in the crossed pyramidal tract was well marked he found degeneration much less in amount, but in the same position, in the other half of the cord (side of lesion). Horsley and Schäfer have, so to say, completed the "motor region" by their discovery of trunk centres in the marginal convolution.

method, into the right lateral column.[13] The degeneration in the (right) lateral column in cases of (left) hemiplegia is recognised by Charcot. So we see that the interconnection of the middle and lowest motor centres is very complex.

Some years ago[14] I inferred from the then known connections of the right corpus striatum (internal capsule) with both halves of the cord (by the first and second set of fibres) that both sides of the body are represented in the right half of the brain (I still say "right" for convenience), but the degenerated fibres being of different columns (the left lateral and the right anterior), that the left and right sides of the body were differently represented in the right half of the brain. (This, I have since stated, seems to me to be but an expansion and modification of the principle of Broadbent's well-known hypothesis as to the double representation of the bilaterally acting muscles.) I should have thought the discovery of the third set of fibres rendered my hypothesis more tenable. But there are serious difficulties. The second set of fibres (those found in the right anterior column) are supposed by some great neurologists to cross to the left half of the cord.[15] The same has been said of the third set of fibres found in the right lateral column. So that it may be that all three sets of fibres pass from the right middle to the left lowest motor centres. It is, however, not really known where the second and third sets of fibres end. No wasted fibres are found in the anterior commissures. Hence, I provisionally keep to the hypothesis mentioned, and now say regarding it that the three sets of fibres may show that the right middle motor centres are connected with lowest motor centres of the left and right halves of the lowest level, and thus that the right middle motor centres represent, by intermediation of the twin lowest motor centres, movements of muscles of both sides of the body. The expression "movements of muscles" introduces a matter which must be considered before I can illustrate the hypothesis.

Having considered the whole central nervous system with the rest of the body represented by it to be a sensori motor mechanism, I now wish to urge that the motor centres of every level represent movements of muscles, not muscles in their individual character. The same muscles, that is, all the muscles, are represented in Simplest, in Complex, and in Most Complex movements by respectively the lowest, middle, and highest motor (better "movement") centres. If so, it is possible, as I believe happens in hemiplegia, to lose one series of movements of muscles, and to retain another series of movements of the very same muscles. Thus, to take a case of left hemiplegia in which the right middle motor centres, if not destroyed, are cut off from lowest motor centres (to neglect the second and third sets of fibres), the condition is not properly described as "loss of power of the muscles" of the left arm and leg, but as loss of complex movements of the muscles of

[13] They were called by Sherrington "re-crossed fibres;" but he now thinks (*Journal of Physiology*, January, 1890) that this name is unsuitable. Sherrington, in a case of small and superficial lesion of one-half of the brain at the lower end of the fissure of Rolando has traced degenerated fibres into both halves of the pons and medulla.

[14] *Med Times and Gaz*, October 23rd, 1869. In the abstract of one of my Gulstonian Lectures (1869), after reference to Broadbent's hypothesis, there appears: "Taking one side of the brain, the right, the lecturer thinks the muscles acting unilaterally, both of the left and of the right side of the body, are represented in the right side of the brain, but that the muscles of the left side of the body are especially represented there. 1st. More in quantity, for they are more affected when the hemisphere discharges. 2ndly. First in time (instability), for they are affected before those of the right side."

[15] Gowers, *Diseases of Nervous System*, vol. i, p. 114.

those limbs. The simplest movements of the very same muscles remain represented by the left lowest motor centres. The muscles certainly are unaffected, and when rigidity comes on the simplest movements of them are developed by over-activity of their lowest motor centres. To repeat, there are, in cases of hemiplegia with rigidity, the double opposites, loss of complex movements and over-development of simplest movements of the very same muscles. The fixed rigid state of the left arm and leg, which clinically we speak of as a symptom, as if it were something *sui generis*, is only the algebraical sum of the co-operating and antagonising "pulls" of the simplest movements of the muscles of these limbs. If the hemiplegia be very slight, we may say that the muscles of the left arm and leg "are only a little weak," but the strict description is that there is then loss of but a very few complex movements of all the muscles of these limbs, with persistence of the rest of the complex and of all of the simplest movements of those same muscles. Similarly, convulsion is not to be looked on as convulsion of muscles; in an epileptiform seizure the convulsion is a contention of complex, and also of simplest, movements. In this contention the individuality of each movement is lost. I will consider a more complex case, that of a man imperfectly hemiplegic permanently, and yet subject to occasional epileptiform convulsion of the region paralysed; the paralysis is loss of but some of the complex movements of all the muscles of the arm and leg, and there is persistence of the whole of the simplest movements of all those muscles; when the fit comes there is a temporary contention of the remaining complex movements and of the simplest movements of all those muscles.[16]

The distinction between muscles and movements of muscles is exceedingly important all over the field of neurology; I think the current doctrine of "abrupt" localisation would not be so much in favour if it were made. The occurrence of convulsion of a muscular region which is already imperfectly and yet permanently paralysed, is unintelligible without that distinction. And without it we shall not understand how it can happen that there is loss of some movements of a muscular region without obvious disability in that region. This bears closely on the realistic study of the physical conditions in aphasia and insanity. We are concerned with both these morbid affections in this inquiry. The anatomical loss in cases of aphasia is of certain complex movements of the tongue, palate, etc.; there is a paralysis in that sense. In cases of insanity (post-epileptic states, for example) there is, I submit, corresponding to the negative element of the psychical symptomatology, paralysis in the sense of loss of some of the most complex combinations of impressions or of most complex movements, or of both; for one example of those most complex, etc., movements of the hands, which are represented in the anatomical substrata of tactual ideas. In brief, a negative lesion of any part of the nervous system ("organ of mind" included) causes, and always causes, paralysis in the sense spoken of, sensory or motor, or both, and causes nothing whatever else.

It may seem absurd to say that when there is no obvious disability in a muscular region, there may be loss of some move-

[16] The distinction between movements and convulsion is of extreme importance, and is not always made. In some slight epileptic fits there are mixed up, so to say, with convulsion movements properly so called, as those of chewing, spitting, clutching the throat, etc. It is very necessary not to mistake writhing movements of the arms with suspended respiration for convulsion of those limbs. I regret that time will not allow me to consider this part of my subject, for the question raised bears closely on the interpretation of post-epileptic mania, and thus indirectly on the study of insanity in general.

ments of that region. This is, however, what I do affirm confidently. We shall be particularly concerned with this dictum when dealing with post-epileptiform aphasia. An eminent physician, referring to a previous statement I made[17] of the dictum, described my aim as being to prove two things — that a motor centre does and also that it does not represent movements. I never consciously attempted that very marvellous feat. Then, as now, I am content with the supposition that the non-disability of a muscular region when some movements of it are lost by destruction of part of a centre is accounted for by that muscular region being represented by other movements in other parts of the centre or in other centres. On this supposition of Compensation, we can explain recovery from hemiplegia (without relying altogether on the hypotheses of subsidence of "shock" or diminished pressure) dependent on very small destructive lesions; there is recovery when a few movements are permanently lost. That the seeming paradox holds for some cases is undeniable, as the experiments of Semon and Horsley on the cortical representation of the vocal cords show. These experiments give a crucial verification of Broadbent's well-known hypothesis for one case. Moreover, France[18] has found degeneration in the lateral column of the cord after lesions of the gyrus fornicatus in monkeys. This shows, by the way, that that gyrus, although no doubt (Horsley and Schäfer) mainly sensory, is not purely sensory. Although in the monkeys mutilated as mentioned, there is no discoverable disability in any muscular region, I submit that the degeneration France describes is proof of loss of some movements, skeletal or visceral, or possibly of negative movements (inhibitory). It would be remarkable if there were any conspicuous disability, considering the enormous compensation given by the intact "motor region." I can now return to the question of representation of both sides of the body in each half of the brain.

I suppose that the right "motor region" represents complex movements of muscles of both sides of the body (1) as they serve in bilateral actions, (2) as they serve in alternate actions (or, I should say, with preponderance of activity on one side), and (3) as they serve in unilateral actions. But I suppose, too, that those movements which are "bilateral" are represented most nearly equally in right and left halves; that those which are "alternate" are represented less equally in the two halves (those of the left side more in the right half), and that those which are "unilateral" are represented most unequally in the two halves (those of the left side most in the right half). We must bear in mind that movements are spoken of, for the so-called alternate muscles may serve in bilateral movements; they may serve with a great degree of preponderance on one side approaching unilateral movement; and the most unilateral muscles may serve in bilateral movements.

It may, however, be asked, Where is the paralysis corresponding to abrogation of the second and third sets of fibres from the right "motor region?" The existence of some weakness of the right side (the so-called non-paralysed side) has been urged by Brown-Séquard, Pitres, Friedlander, and Gowers; but when in the difficult circumstances attending investigation of the state of the right side no disability is demonstrable, there may yet be loss of some few movements of the muscles of that side. The loss of these few movements is masked by the compensation given by the

[17] JOURNAL, May 10th, 1873.
[18] *Phil. Trans.*, B. 41, 1889.

left half of the brain, which represents most numerous movements of the right side. If we take the case of excessive discharge beginning in some part of the right middle motor centres, we find that, in severe epileptiform fits, both sides of the body are convulsed. In this case compensation is "inverted," that is to say, centres which would compensate a loss are compelled to co-operate in an excess. The comparison and contrast of hemiplegia and an epileptiform seizure are, however, by no means simple, as we shall see when the process of universalisation of an epileptiform convulsion is considered.

Although when speaking of the hierarchy of nervous centres I excluded from consideration the higher divisions of the cerebellar system, it is necessary to say something of them. There are in some cases of tumour of the median lobe of the cerebellum, seizures very like, if not quite like, those of ordinary surgical tetanus. I have suggested that the convulsion of surgical tetanus is owing to cerebellar discharges; but no doubt the poison causing that morbid affection affects lowest centres too. It has been suggested that the tetanus-like seizures in the cases of cerebellar tumour mentioned, are owing to pressure upon, or to changes induced in, the corpora quadrigemina or medulla oblongata, or both. But surgical tetanus is, at all events, in order of development of spasm over its regional distribution the "complementary inverse" of epileptiform seizures. And when we consider nervous diseases, each as a flaw in a whole nervous system, the study of "corresponding opposites" is most important. There is a more direct reason for taking note of the cerebellum in the present inquiry. What the structures are connecting the cerebellum with the lowest motor centres I know not. If there be a connection—if the lowest level is at once the lowest level of the cerebral and of the cerebellar system—we may expect the higher levels of the cerebellar system to be concerned in post-epileptiform states; the question is important, especially with regard to increased tendon reactions in post-epileptiform paralyses. I have suggested[19] that the cerebellum is concerned with the rigidity and exaggerated tendon reactions of (left) hemiplegia[20] when that paralysis is the result of a destructive lesion of the (right) internal capsule, that there is not only exaltation of function of the (left) lowest motor centres from loss of control, but also that the cerebellar influx upon them is no longer antagonised. This hypothesis has been objected to on the ground that in transverse lesions of the upper dorsal or cervical cord (the cerebellum being thus excluded) there is nevertheless rigidity of the legs with exaggerated knee-jerk and foot clonus. I was obliged to admit that the cerebellum is not necessary for the production of the exaggerated jerk and rigidity.[21] But Charlton Bastian,[22] who has advanced proof where I only speculated, has found that, in man, on complete transverse lesion of the regions of the cord mentioned, the muscles of the legs are flabby and the knee-jerks absent; what is very striking, he finds that the condition of rigidity of the legs and exaggerated knee-jerk in cases of incomplete transverse lesions changes to one of flaccidity with no tendon reactions when that lesion becomes completely transverse. If Bastian be correct, as I think he is, the hypothesis of cerebellar influx, on which we are in fundamental agreement, is supported.

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[19] *Medical Examiner*, April 5th, 1877, and March 29th, 1878.

[20] Not rarely both knee-jerks are exaggerated; this perhaps helps to show that the third set of fibres ends in the right lowest motor centres.

[21] *Medical Times and Gazette*, February 12th, 1881.

[22] Quain's *Dictionary of Medicine*, p. 1481.

Lecture II.

Epileptiform (Middle Level) Fits.—Varieties.—Diluted Convulsions.—Crude Sensations.—Locality and Order of Convulsive Movements.—Degrees and Ranges of Fits.—Hyper- and Hypophysiological States.—A Discharging Lesion.—The "Buttoning Centre."—Physiological Fulminate—The March of the Convulsion.—Compound Order.—Cells of a Discharging Lesion Quasi-parasitical.—Nutrition of Cells.—Summary.

I TAKE for most particular consideration the second kind of fits, epileptiform seizures (middle level fits); they were first described by Bravais in 1824. There are many reasons why we should study them first. (1) Their investigation is comparatively easy. We can, if present at a paroxysm, ascertain the place of onset and trace the March of the convulsion; and if we witness no attacks, since consciousness is not lost or is lost late in the seizures, the patient can tell us the place of onset and much more about his fits. (2) They have permanent paralytic counterparts in monoplegias and hemiplegias from destructive lesions of the "motor region." (3) There is after them, at least in many cases, temporary paralysis of the parts first and most convulsed. (4) There is not rarely permanent paralysis of the parts which are occasionally convulsed, this showing—a very important thing with regard to doctrines of localisation—that in the same case there is coexistence of a destructive lesion and a discharging lesion of different elements of the same centre. (5) Because there is after some seizures temporary defect of speech ("partial aphasia"). (6) Because, if we can find the condition of centres answering to paralysis and aphasia after these seizures, we shall be greatly helped in our investigation of the state of the highest centres in insanities after epileptic fits. (7) Because epileptiform seizures often depend on gross organic disease, such as tumour (syphilitic or non-syphilitic); hence, we may at *post-mortem* examinations obtain proof or disproof of any notions on localisation and gross pathology we had from a study of the patient's symptomatology during his life. (8) It is in epileptiform seizures that operations have been done by Macewen, Godlee, Horsley, Barker, and others. Hence, very precise study of fits of this kind is necessary. In this regard, as well as in many others, the minute investigations of the "motor region" by Beevor and Horsley are of great value.

It would be very unmethodical to begin the scientific study of the more complex (epileptic) kind of fits before the study of the epileptiform kind.

There are Varieties of epileptiform seizures. The convulsion begins in some part of one side of the body. Varieties are distinguished by the particular place of onset ("signal symptom" of Seguin) of the convulsion. The fit may begin in some part of either right or left side of the body; I call that on which it does begin the "first side," and the corresponding (opposite) half of the brain in which the discharge begins the "first half." If the fit begins on the left side, that is the first side, and the right half of the brain is the first half; suppose that the convulsion becomes universal, then the right side of the body is the second side, and the left half of the brain (if it discharges after the right) is the second

half. Whether the onset be right or left sided is a very important matter, because there is often defect of speech after right-side-beginning fits; moreover, on account of the "speech centre," operations on the left half of the brain are more serious than operations on the right.

The three commonest Varieties of epileptiform seizures are—(1) Fits starting in the hand (most often in the thumb or index finger or in both). (2) Fits starting in one side of the face (most often near the mouth) or in the tongue, or in both these parts. (3) Fits starting in the foot (nearly always in the great toe). (Of course one always means convulsive development of movements of muscles belonging to the several parts mentioned.)

Of necessity the three Varieties depend on the fact that the local discharging lesion is of cells of different parts of the "motor region"—of hand, face, and foot centre respectively. The starting point is almost invariably the same in each patient, but not always. A patient whose fits commonly begin in the hand may sometimes have the face of the same side slightly and solely affected; or a patient may tell us that his fits "fly about"—that is, leaving the face for the hand, or *vice versâ*.

We sometimes meet with paroxysms of one-sided tremor—what looks superficially like tremor, really a "diluted convulsion"—dependent, I think, on discharges beginning in motor elements of the so-called sensory centres behind the motor region.[23] I do not call these epileptiform seizures.

Often enough there is "tingling" or some other crude sensation in the place of onset before convulsion starts; this is part of the proof that the so-called motor region, although mainly motor,[24] is not purely motor. Sometimes there is at or near to the onset of an otherwise ordinary epileptiform seizure excessive development of colour, or of sound; these also are Crude Sensations. Epileptiform seizures with crude sensations deserve very careful attention, but time will not allow me to consider these complications.[25]

It is worth mentioning that some patients have a feeling as if a part were convulsed when it does not really move; one of my patients subject to veritable convulsions beginning in his left thumb had sometimes what he called "convulsions not to be seen" of that part. Another patient had the feeling of convulsion of one side of his face, but looking in a glass he saw that it did not move. He died of brain tumour (necropsy), but its exact position I did not learn. A patient who has fits starting in the hand will say that he feels "it" in the face, when, however, the face does not move; this "it" may be owing to very slight discharges of sensory or motor elements, or of both.[26]

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[23] I have never believed that any part of the cortex is purely motor or purely sensory. If the elaborate visual projections in some cases of migraine occur during discharge beginning in a "sensory centre," then the elements of that so-called sensory centre are only chiefly sensory; the fortification outline and the vibrations of parts of the migrainous visual spectre imply discharge of motor elements as certainly as the colours imply discharge of sensory elements.

[24] See remarks (Lecture I) on France's *Researches on Degeneration of Fibres of the Lateral Column of the Cord, consequent on Lesions of the Gyrus Fornicatus.*

[25] Of course crude sensations (psychical) and convulsion (physical) are in no way comparable: the comparisons and contrasts are of excessive discharges of sensory elements *during which* crude sensations arise, with such discharges of motor elements *from which* convulsion arises.

[26] When a healthy person thinks of doing something ("has an idea of a movement"), I consider that what occurs physically is slight discharge not only of sensory but also of motor nervous arrangements of his highest centres; there is "nascent movement"—slight discharge of the very same motor nervous arrangements of the highest centres which, if more strongly discharged, would, by intermediation of middle and lowest motor centres, produce the actual movement. It is probable that a similar explanation (the middle motor centres

Epileptiform fits begin in the Animal parts of the body, and commonly in those of them which are the most animal. Thus the majority begin in the arm, and nearly all these in the most animal part of that most animal part, the hand, and most of them in the thumb or index finger or both—the most animal parts of the whole organism. Perhaps the term animal is awkward in this connection; it is convenient in contrast to organic. We can, however, use other terms, and say that epileptiform seizures most often begin in those parts which, speaking popularly, have the most "voluntary" uses; in those parts which have great independence of movement; in those parts which have the greater number of different and more special (definite) movements, at the greater number of different intervals. The foot in man has, however, few *different* movements; the hallux in him is much less specialised than in the monkey. It is, then, a very striking thing that epileptiform seizures beginning in the leg almost invariably start by spasm of the great toe, in the most, and yet but little, differentiated part of the whole lower limb.

It may be objected to the principle stated that, to take an example, convulsion in an epileptiform seizure may begin in the shoulder, the least "voluntary" part of the arm, and spread down the limb [27] I have considered this objection in the JOURNAL of August 17th, 1889. We have, as I continually insist, in strictness to speak not of representation of parts of the body (muscular regions), but of representation of movements. Movement of any part of the body done with intention is a "voluntary" movement. Evidently when a card-sharper shrugs his shoulder as a sign to a confederate to play a trump, that is his then most voluntary movement. In so far as any part of the body has a movement independent of the rest, in so far that movement has, I suggest, a degree of localness of representation in the middle and highest motor centres. Horsley and Beevor assign a point of the "motor region" for the primary movement of each segment of the upper limb. It may, however, be taken that epileptiform seizures most often begin in the most "voluntary" parts, and that admitting many varieties the three commonest are selected for comment.

Epileptiform seizures illustrate Dissolution—dissolution in process of being effected—in which the order is from "voluntary" towards automatic. *In normal development of movements the order is the opposite—is that of Evolution; it is from automatic to "voluntary."* [28]

The "motor region" (motor province of the middle level) presumably represents at least motorily the whole of the body, demonstrably nearly all parts organic as well as animal. Hence, as the discharging lesion may theoretically be of any part of the "motor region," it is a legitimate hypothesis that there are fits starting by excessive discharges of cells of nervous arrangements of the "motor region" which especially represent parts in the organic field, and that some fits called *epileptic* are such seizures. I do not hold that hypothesis. There are parts—for example, the vocal cords—which we may speak of as organico-animal.

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being here primarily concerned) applies to the "ideas of convulsion" spoken of in the text. No doubt if a man subject to fits beginning in his right thumb were to lose the right arm by amputation his fits would still *seem* to him to begin in his thumb—to begin in his spectral thumb, for some time at least.

[27] I have recorded a case of this kind, *Medical Times and Gazette*, June 5th, 1875.

[28] Properly from most automatic to least automatic in evolution; the opposite in dissolution. "Most voluntary movement" is objectionable because it is a mixture of psychological and anatomico-physiological language; it is a popular expression, equivalent to what is scientifically "least automatic movement."

Munk and Krause[29] in the dog, and Horsley and Semon in the monkey, have investigated the cortical representation of the movements of the vocal cords. Krause in the dog, Horsley and Semon in the monkey,[30] produced convulsive seizures by excitation of the cortical "laryngeal centre." Semon thinks that laryngismus stridulus in man (the infant human being) is a cortical fit; but if so there is not an exception to the statement that epileptiform fits begin in the animal parts, if we adopt Semon's views of, and inferences from, the kind of representation of the vocal cords in the motor cortex ("purposive or volitional, since it is adduction, that is phonation").

Although epileptiform seizures most often begin in the three most animal parts of the animal parts mentioned, yet as the fits go on the organic field becomes involved. In a dog poisoned by curara (respiration being artificially kept up) the animal parts are excluded, and then we see that by excessive discharges experimentally induced in its "motor region," effects are produced in, and are then limited to, the organic field.

There are Degrees of each variety corresponding to degrees of the "severity" of the excessive discharge. Degrees are to be considered with respect to two factors, (1) amount of convulsion, and (2) range of convulsion. I illustrate here quite arbitrarily by Range only, and take the case of an epileptiform fit starting in the thumb. Admitting that there are many ranges, it is allowable to make arbitrary divisions (they must not be taken for real distinctions) into four Ranges: (1) Terminal[31] fit—the spasm involves, say the hand, or some part of it only; (2) monospasm—the arm becomes involved; (3) hemispasm; (4) bilateral convulsion—the second side being gained, the fit becomes universal. Observe the use of the word "become." In the range (4) there is a March of spasm from the part first seized all over the body, answering to increased spreading of the central excessive discharge beginning in some particular part of the motor region of the first half, and may be extending to the second half, of the brain. The second side of the body (in 4) is affected later, and commonly less than, the first side.

From (1) to (4) there is but a single variety of epileptiform seizure in four different degrees. Otherwise stated, a man who has had convulsion of the range (1) or (2) does not become subject to another variety of epileptiform seizure when he has also fits which, beginning in the same terminal part (hand, we are supposing), have a more extensive range however much more extensive it is; so to speak, that patient is subject to *a* fit, and has it in different degrees on different occasions. However limited or however wide the range of convulsion, the corresponding discharge begins in the very same centre of the motor region of but one, the opposite half of the brain. This means that it is the place of onset ("the signal symptom" which localises)[32]: it points to the par-

[29] *Sitzungsberichte der Königlichen Academie der Wissenschaften und Archiv für Anatomie und Physiologie*, 1883.

[30] JOURNAL, December 21 t, 1889.

[31] It may be well said that a fit beginning by spasm of the cheek does not begin in a *terminal* part; but the meaning of the word terminal may, for my present purpose, be used to include that onset. Peripheral would have too wide a meaning. I would suggest acro-epilepsy as a name for epileptiform seizures were it not that it might mislead some to think that there are epileptiform seizures which begin in the nose.

[32] The term aura is, I suppose, never used now in its original sense. When a medical man speaks of an aura in an epileptic fit starting from the epigastric region, he is understood to mean that some sensation is referred by the patient to that part of the body, and to believe that the sensation arises during the incipience of a discharge of some central sensory elements.

ticular part of the "motor region" of the cells of which part the discharging lesion is made up. When a man's epileptiform fits "get worse"—when we are supposing they become of greater range—there is the same discharging lesion, but it has become of more cells, or the cells of it have become still more highly unstable, or perhaps there are both kinds of change; in consequence of this purely local ingravescence, the induced discharges (compelled discharges) of normal stable cells are more numerous.

Were A—and B—to be universally convulsed, they would have different varieties of epileptiform seizures if in A—the onset was by spasm of the hand, and in B—by spasm of the foot, however much alike the fits looked at the acme of each; the discharging lesion in A—would be of some cells of his "arm centre," in B—of his "leg centre." To note the "signal symptom"—to use Seguin's term again, and therefrom to infer the seat of the discharging lesion—is more important, obviously for "brain surgery," than anything else about the paroxysms.

It has been implied, but it may be well to say explicitly, that an epileptiform seizure does not, when become universal, turn into the other *kind* of fit—the epileptic. I mention this because I think a rapidly universal epileptiform seizure superficially resembles an epileptic one, and a slowly developed epileptic seizure superficially resembles an epileptiform seizure.

I have often urged that the Clinical Problem in every nervous malady is of three elements: (1) Anatomical, (2) Physiological, (3) Pathological. In other words, we have in every case to seek: (1) the seat of the lesion, the structure damaged, (2) to infer the kind of functional change of structure, and (3) to discover the nature of the abnormal nutritive process of which the alteration of composition of nervous matter producing the abnormal functional state of structure is effected (*vide infra*).

It may be properly said that structure, function, and nutrition never exist separately. It is quite obvious, taking for comment normal conditions, that there would be no persistence of function without continuous nutrition; that function is not conceivable apart from some structure, and that structure without function is nothing for an organism. It is, however, convenient, if not necessary, for clear exposition of nervous maladies, to consider for a time each of the three separately. It will, however, be impossible to consider one without frequent explicit and always implicit reference to the others. Perhaps I use the term Anatomy regarding the nervous system in an unusual sense; I do not use it convertibly with Morphology. A knowledge of the Anatomy of any Centre is *a knowledge of the parts of the body which that centre represents, and of the ways in which it represents them.*

Anatomy.—(Localisation.) I shall say very little under this head. The patient A. is subject to fits, every one of which begins in his right thumb; there is a lesion of the thumb centre of the left middle level ("motor region")—this is the anatomical diagnosis. (According to Beevor and Horsley, "the middle third of the ascending parietal convolution is the focus of representation of the movements of the thumb.")

Physiology.—Physiology deals with the dynamics of the organism—that is, with its function. I use the term "function" with regard to nervous diseases in a strict sense, and never in the way it or its adjective is used when applied to the symptoms of a hysterical woman, or to minute or transitory changes of structure.[33] I must now define the term. The function of organic

[33] See an able paper by Dr. Allchin, *Westminster Hospital Reports*, vol. ii.

matter and *par excellence* of nervous matter, to which I confine further remarks, is to store up energy,[34] to liberate that energy (nervous discharge) at different rates against resistances of different amounts; the energy liberated is dissipated or does work, or there are both consequences according as the resistances encountered are or are not overcome, or are only partially overcome. We have here to deal with abnormalities of this function—that is, with abnormal physiological states (I need not always add the adjective abnormal).

There are two kinds of physiological or functional states in cases of disease of the nervous system. (1) Function may be exalted, and is, sometimes, as that of cells of a discharging lesion, very greatly exalted; these are superpositive functional changes—that is, hyper-physiological states. (2) Function may be diminished or lost; these are negative physiological or functional states—they are hypo-physiological states. Destructive lesions come in this category, although there is in them loss, not merely of function, but of functionable material also.

The two kinds of functional changes—the hyper- and the hypo-physiological—are opposites; there are no degrees from one to the other; so to say, they depart from normal function, the one upwards, the other downwards. We shall have to speak later of a negative functional change in temporary post-epileptiform paralysis; that negative change (there is temporary exhaustion of nervous elements) is a direct result of the excessive discharge in the prior paroxysm; the two opposite functional states of the same nervous elements occur in immediate sequence.

Now I consider the inclusion of negative functional states in cases of mere temporary exhaustion of nervous elements in the same category with destructive lesions when nervous elements and function are gone together. I dare say the former would be called functional, because the alteration in the composition of the material of nervous elements, whatever its nature, is no doubt slight and is certainly temporary. But I call it a negative functional change, *because function is lost:* the slight and temporary alteration of composition is a pathological change. For the time being fibres exhausted are not in effect nerve fibres at all; with a morphological plenum there is a functional vacuum as certainly as there is when they are destroyed. Whether nervous elements are functionless or gone altogether, the situation *for the time being* is the same. I shall speak of both as "negative lesions." And saying that the exhausted fibres recover soon is recognising fully the vast difference in their condition from that of fibres destroyed —properly, absence of fibres.

There are degrees of negative function from defect to loss. Of these I will say nothing. There are two greatly different degrees, if indeed we may not say two kinds, of superpositive (hyper-physiological) function. We are concerned with both in this inquiry; it is imperative to distinguish them clearly.

There is (*a*) the vast exaltation of function of cells of a discharging lesion; this degree is always produced by some pathological process—that is, by morbid nutrition, a nutrition which alters the composition of nervous matter.[35] This, the first degree of func-

[34] Assimilation of material having potential energy (anabolism); decomposition of material (katabolism) with liberation of energy.

[35] This is the definition I would suggest of a pathological process (morbid nutrition) or of its result. I suppose that such poisons as strychnine alter the composition of nervous elements, in consequence of which morbid nutrition (a substitution nutrition) there is a functional change of the degree (*a*). It seems more likely that strychnine enters into the very composition of matter of nerve cells than that it stands outside the cells and "irritates" them. When a

tional change, is of a highly explosive character; it issues in paroxysmal discharges.

There is (*b*) a comparatively slight exaltation of function with which we shall be particularly concerned when we consider post-epileptiform conditions. In these conditions, besides paralysis answering to a negative functional state of fibres of the second segment of the kinetic route, there are increased tendon reactions answering to a hyperphysiological state of this degree (*b*) of cells of anterior horns (lowest motor centres). I believe that the second degree of superpositive functional state is not—certainly not in the case just alluded to—the direct result of a pathological process, but that it is the *indirect* result of a negative functional state of *other*, related nervous elements. (Here is, essentially, an application of a principle long ago put forward by Anstie and Thompson Dickson). This, the second degree of functional change, does not issue in paroxysmal discharges, but in continuous discharges; or discharge can at any time be evoked by appropriate slight excitations. Returning to post-epileptiform conditions, the negative functional state answering to the paralysis is alone produced by a pathological process; the abnormal condition of anterior horns implied by increase of the tendon reactions is one of over-activity of perfectly healthy nervous elements. There is simply the proper activity of certain lowest motor centres, which is manifested at its maximum when an obstacle has been removed, when control has been taken off. I think, too, that cerebellar influx is no longer antagonised (see Lecture I); and if so, the cerebellum is simply unhindered in its activity, and is doing, now that an obstacle is removed, what it was always "trying to do." Here, again, we come across the principle that parts of the symptomatologies of nervous maladies are owing to activities or over-activities of perfectly healthy nervous arrangements. If this principle be valid, it is evidently important to distinguish between the physiological and the pathological factor in nervous maladies. Of course increased activity of any nervous elements implies increased nutrition, but there is not, I submit, in the case of elements answering to the second kind of functional change, morbid nutrition. There is not, then, what I call a pathological process; the composition of the matter of the cells concerned is not altered. For the present I shall speak only of the first degree (*a*) of superpositive functional states—that which is of a highly explosive degree—and is produced by a pathological process.

The hyperphysiological state (degree *a*) in the case of A—(*vide supra*) is what I call a "discharging lesion." It is a crude hyperphysiological state just as its diametrical opposite, loss of function, is a crude hypophysiological state. I continue to speak of healthy cells as stable; they are naturally unstable, of course, but I mean by using the term stable that they are so *in comparison with* those of discharging lesions, which I speak of as being *highly* unstable. The discharging lesion is of a few cells which have got far above the rest of the cortical cells in degree of tension and instability of equilibrium. That lesion is made up of cells of nervous arrangements which represent some most special movements of a particular muscular region; the sudden and excessive development of these movements from discharge of those cells is

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rabbit's respiratory centres discharge excessively on withdrawal of blood, one must suppose that before that discharge there is an abnormal metabolism (pathological process) whereby matter of the cells of the centre becomes more explosive. Certain "pathological processes," such as cerebral hæmorrhage, are really injuries; hemiplegia resulting from clot is, so far as destruction of fibres goes, like that caused by the prong of a pitchfork; there is no real pathological process in either of these modes of destruction.

the convulsion incipient ("signal symptom" of Seguin), or, if there be no spreading of the spasm, it is the convulsion total.

The hyperphysiology of epileptiform seizures is the element of the threefold clinical problem of which we are sure. We are quite certain that normal movements are the results of liberation of energy attending katabolism; thus the inference is irresistible that that sudden and excessive development of many movements at once, which we call convulsion, must be a result of an excess of the same physiological process—of a sudden and excessive energy-liberation attending great and rapid katabolism. I will give illustrations of the different effects of discharges of stable cells (cells of comparatively low tension and of comparatively stable equilibrium) in normal operations, and of those of cells of a discharging lesion (cells of very high tension and very unstable equilibrium) in a convulsion.

It would be most absurd to say that there is a Buttoning Centre, but for convenience of illustration we may imagine such a centre. I illustrate by motor elements only, although, of course, sensory elements are concerned in all operations. In the operation of buttoning there are slight and slow discharges of normal stable cells (fibres are understood) of different nervous arrangements of our supposed centre (lower centres and muscles being understood) simultaneously and successively; in consequence there is a harmony and a melody of the different movements of the hand and arm by which the button is got into its hole. There is Harmony by Contemperation[36] of different movements, and there is Melody in that the compound contemperated movements follow one another at proper time intervals; there is what is commonly called co-ordination, although the time element (melody) is not much considered in most accounts of the process of co-ordination. So much for the discharges of stable cells in the operations of health.

Now let us suppose that the cells of the buttoning centre become by some, any, pathological process, so highly unstable as to constitute a discharging lesion. (For the present we shall speak as if the excessive discharges of the highly unstable cells did not provoke similar discharges of stable cells of collateral centres.) The discharges are sudden and rapid,[37] and of all the cells nearly at once; they do not cause a speedy and vigorous act of buttoning, but a short, severe, and rapid contention of all the movements of that operation; this contention is convulsion of the hand and arm. There is no harmony and melody; on the contrary, in the tonic stage, all contemplation is lost and all time-intervals are merged, so that there is but one rigid state of the musculature of the hand and arm, and, in the clonic stage, there is but a succession of such rigid states. There are no movements properly so-called in this convulsion; but, if I may use the word for once, there is first but a single big useless movement, and next a series of such so-called movements which do nothing but "mark time."

The illustration, although the supposition of a buttoning centre is grotesque, may render clear what presumably occurs when A— is "attacked by his convulsion." The convulsion is a brutal development of that man's own movements. I make this odd remark because I do not think that it is always vividly realised

[36] Contemperation is, says the *Imperial Dictionary*, an obsolete word. One of its meanings given *op. cit.* is, "The act of reducing a quality by admixture of the contrary."

[37] I do not mean by using the word rapid, here or anywhere, that nerve impulses travel more rapidly in the case of excessive discharges, but that more occur in a given time than is normal—that there is a greater quantity of motion in a given time.

that an ordinary severe epileptic "attack" (to take that kind of fit for a moment's illustration) is nothing more than a sudden excessive and temporary contention of very many of the patient's familiar normal movements—those of smiling, masticating, articulating, singing, manipulating, etc. A convulsion is not something altogether *sui generis*. Speaking figuratively and more generally, and still of an epileptic paroxysm, there is the mad endeavour of the highest centres to develop the maximum of function of every part of the body, animal and organic, and of all parts at once; the phenomena of a very severe epileptic fit show that this endeavour is nearly successful; the patient is almost killed by the paroxysm, and is nearly dead (deeply comatose) after it.

The cells of a discharging lesion are not to be thought of only as occasionally discharging excessively; we have also to consider their aspect of too easy dischargeability, otherwise their highly unstable equilibrium. In health the cells of the "buttoning centre" are made to discharge slightly on special excitations of definite force, so that their discharges are in particular relations, and in some degree of community with those of cells of other centres (I will call them "collateral stable cells") with which they are connected. The cells of the "buttoning centre," when they have become highly explosive, are still integral parts of the nervous system, and have the very same connections they had before some pathological process so altered their nutrition that they became highly explosive (and then, metaphorically speaking, "mad parts"). Their equilibrium can be upset, they can be made to discharge, and to discharge excessively, by less special excitations of less definite force coming to them from collateral stable cells—that is, their discharge depends on their own easy dischargeability rather than on the particularity of the excitation reaching them. Possibly their discharge is spontaneous when tension is very great, and equilibrium very highly unstable.

I repeat that the cells which have become very highly "explosive" (those of the discharging lesion) continue to be elements of the same nervous arrangements, and that these nervous arrangements are still connected in the very same ways with the remainder of the nervous system[38] as when their cells were stable. Hence I do not speak of the highly explosive cells as being *in* any motor centre, but as being *of* that centre. I submit that the highly "explosive" cells of a discharging lesion will on their fulminating discharge overcome the resistance of, and thus produce excessive discharge of, collateral stable nervous elements. The epileptiform fit (excepting, perhaps, some slight terminal fits) is not the result only of the discharge of the cells of the discharging lesion, as was supposed for a limited illustration, when speaking of the imaginary "buttoning centre." Making a purely arbitrary limitation, we may imagine that the primary excessive discharge, that of the fulminate of A.'s thumb-centre, only produces directly the initial spasm, that of his thumb (the "signal symptom"); all the rest of his convulsion will be indirectly produced by it, by *compelled* excessive discharges of stable cells. Of these compelled discharges I shall speak again later.

It will now be seen why the term physiological fulminate is occasionally used. I use it in almost a literal sense; the discharging lesion is supposed to be a detonator of collateral stable cells, just as a fulminate (in the artillerist's use of the term) is of the

[38] There is the obvious qualification that some nervous elements may have been destroyed by the same pathological process which caused high instability of other elements, and thus that some connections of the latter are cut.

comparatively stable gunpowder in a cannon. I believe that the only thing persistently physiologically abnormal in A. is that some few cells of his thumb-centre have become fulminant. To speak figuratively, this "mad part" compels many collateral "sane" cells and cells of middle and lowest motor centres, and ultimately the muscles, to co-operate in its occasional and sudden excesses—makes them "act madly" for a time. If the few highly explosive cells, those of the discharging lesion, could be destroyed, the patient would be rid of his fits; he would lose nervous elements which are doubtless never of value for co-operation with the collateral stable cells in normal operations; he would lose cells of negative value and of positive injury—cells like those of an animal poisoned by strychnine, which, on their discharge, "run up" movements into useless contentions. The cells of A.'s fulminate, when called on to co-operate with normally stable cells in any operation, function excessively, and so as, after the manner of a detonator, to cause wide excessive discharges of many stable cells. It is a pity that A. cannot be rid of these worse than useless cells; but I know of no way of effecting this riddance. There is the surgical question of cutting out part of the cortex.

I have, in the foregoing, used, regarding hyper-physiological discharges, the term excessive, and, regarding the resulting convulsion, the term severe; the more excessive the discharge the severer the fit. I now use more precise terms, and consider this part of the subject as it was in effect, so it seems to me, long ago considered by Herpin. I regret greatly that my ignorance of physics renders me unable to deal with it adequately.

With regard to nervous discharges, or, as I shall here say, liberations of energy by nervous elements, we have to consider two aspects—quantity of energy liberated, and the rate of its liberation; the two varying factors both in normal and in "excessive" nervous discharges. With regard to the convulsion, we have to consider its degree, and the rate with which it is produced.[39] We have to study the amount of convulsion, the range of convulsion, and particularly the "deliberate" or "sudden" rate of onset. In two liberations of equal quantities of energy at different rates there is the same momentum or quantity of motion. But the force of the more rapid but shorter liberation of energy will be greater than that of the slower and longer liberation. Force only exists while it lasts; there is no doctrine of conservation of force. The more rapid the liberation of energy by a discharging lesion the greater resistances will be overcome, the more numerous collateral stable elements will be compelled to discharge, and thus the more the amount of convulsion and the greater its range.

I suppose that there are degrees of instability of the cells of the fulminates in different cases of epileptiform seizures and at different periods of the same case. When A.'s fits "get worse" (greater amount of convulsion and greater range of convulsion), more cells may have then become highly unstable, or those already highly unstable may have become still more so; his fulminate becomes more fulminant, but that fulminate is made up of cells of the same part of the "motor region," if not of the very same cells. I now consider it must be taken hypothetically, the differences of fulmination of discharging lesions. Let me suppose two cases, in each of which there is a discharging lesion constituted by cells of the thumb centre. In the patient A. the cells are (I am only able

[39] Herpin, in his valuable work on *Epilepsy*, sums up several propositions as follows (italics in original):—"En résumé: *plus le début est long, moins la crise est violente, plus il est instantané, plus l'accès est intense.*"

to distinguish vaguely) of lower degree of high instability (his fulminate is much less of a fulminate) than in the case of A. A. In both the first spasm is of the thumb. I shall mention the cortical discharges only; the sequent discharges of the lowest motor centres and of the muscles are to be taken for granted.

In A—the primary discharge, that of the fulminate, is deliberate, produces (secundo-primary) discharges of few collateral stable cells, and produces them slowly one after another; the corresponding convulsion sets in deliberately and spreads slowly; is of little range (very local; for example, of the arm only), is of long duration, and there is an easily traceable distinct sequence over the range (all this, of course, comparatively with what occurs in the next case). In A. A. the primary discharge is sudden, produces (secundo-primary) discharges of many collateral stable cells, and produces them rapidly and more nearly at the same time; the corresponding convulsion sets in suddenly and spreads rapidly; it is of great range, is of short duration, and there is a less easily traceable distinct sequence over the range.

The more rapid, though the shorter, of the two primary liberations by the discharging lesions—supposing an equal quantity of energy to be liberated by each—will overcome greater resistances, and will thus compel discharges of a greater number of collateral stable elements; the convulsion produced will be both of greater amount and of greater range. Lines of many different degrees of resistance will be overcome by such a primary liberation more nearly at the same time; the convulsion will more quickly attain its maximum at every part affected, and will be more nearly of the same degree in all parts affected.

We have not only to note how much of the body is ultimately involved, but also the order in which the several parts involved are affected—the March of the convulsion. There is not a simple, but a compound sequence of spasm; the convulsion does not cease in one part when another is involved. To observe, to give a simple example, how much of the arm has been involved when convulsion appears in the face will, I think, help us to clearer notions of localisation of movements of those two separate parts of the body in the centre discharging (anatomical localisation); or if not, at any rate as to the time relations of different elements of different centres (physiological localisation). From increasing discharge of a motor centre there is a double effect; there is not simply "more convulsion," there is (1) greater amount of convulsion of the part first seized, and there is (2) extension of convulsion to the next part of the same muscular region, or to some other part represented in the centre discharging (or in another centre connected with the one primarily discharging by particular time relations). Most generally, the progress of the two dissolutions—convulsion from increasing discharge of a motor centre, and paralysis from increasing destruction of a motor centre—may be rudely (and without the least pretence at exact quantification) symbolised as (1) x, (2) x^2y, (3) x^3y^2z; the order x, y, z, implies the representation in the centre supposed of the time relations, and of the degree of speciality of movements of the three parts. Horsley and Beevor find Compound Order in their development of "simple movements" by very slight excitations of small parts of a monkey's "motor region." After developing what they call the Primary Movement of a part, say one of the shoulder, there follow, on increasing the excitation, secondary and tertiary movements—that is, there is produced a sequence of movements of segments of the arm. But when the secondary movement comes the primary has not ceased; on the contrary, it is

intensified: the development may be roughly symbolised as p and then p^2s.

Returning to epileptiform seizures, Compound Order is observed on a small scale in fits involving limited regions of the body (very well seen in the face), and on a large scale when such seizures become universal. There is a very intricate compound sequence from the beginning of the fit to its universalisation. I shall, however, make artificial separations, and say a fit which becomes universal that excessive discharge beginning in some cells of a part of the right middle motor centres produces convulsion (1) starting in the left hand and spreading up the arm and down the leg (first side), (2) involving both sides of the trunk, and (3) finally gaining the limbs of the right (second) side when all parts of the body are in convulsion together. That the left limbs are convulsed from discharge of the right middle motor centres, by intermediation of the first set of fibres of the second segment, is not doubted. I used to suggest that convulsion of both sides of the trunk and of the right limbs is also produced by discharge of that half by the second and (or rather as I should now say) third sets of fibres. This was rash, for supposing that some convulsion of the second (right) side *is producible* by discharge of that one half (right), it does not follow that the convulsion which actually occurs *is produced* by it alone. For my hypothesis of representation of both sides of the body in each half of the brain, it would suffice if there were any degree of tonic or clonic convulsion of the right (second) side, for I only suppose that the right side is represented less than and subordinately to the left side, and also second in time in the right half. The subject is a difficult one. Horsley finds that when the corpus callosum is divided, excitation of the right (I continue to say "right" for convenience) middle motor centres produces epileptic convulsion of the left limbs only.[40] In the case of dogs, Franck and Pitres found that artificially induced discharges of the right "motor region" caused universal convulsion when the left motor region had been extirpated and the corpus callosum divided. I quoted them to this effect in my third Croonian Lecture.[41] They, however, as I then said, attribute the universalisation of the convulsion to the pons, medulla, and cord (which together I call the lowest level).

Considering, then, the opinions of those who hold that the second and third sets of fibres, as well as the first set (all the fibres of the second segment of the kinetic route interconnecting the right middle motor centres with the lowest motor centres), end in the left lowest motor centres, the researches of Franck and Pitres and those of Horsley, and also the great complexity of the subject, I ought not to be dogmatic as to the process by which universalisation of an epileptiform convulsion is produced. Most likely in severe seizures the left middle motor centres are discharged, as Horsley supposes, after the right by intermediation of callosal fibres, although possibly some slight convulsion of the right limbs is produced from discharge of the right half alone by intermediation of the second and third sets of fibres. And most likely, too, the anterior commissure of the pons, medulla, and cord (intrinsic fibres of the lowest level interconnecting left and right lowest

[40] In the abstract of "Brown Lectures," *Lancet*, December 25th, 1886, Mr. Victor Horsley is reported: "The conclusions ..... all round, therefore, were very emphatic, to the effect that convulsions due to cortical discharge are evoked in various groups of muscles by nerve energy proceeding from that centre in each hemisphere which is in relation to each group of muscles, and that in generalised epileptic convulsions both cerebral hemispheres are involved.

[41] JOURNAL, March 29th, 1884.

motor centres) are concerned. Probably the loss of consciousness answers to excessive discharges of the highest centres caused by the medium of sensory (upward) fibres. The process of universalisation of epileptiform seizures is a very intricate one, and deserves more precise analysis than I am capable of making.

I stay here to say that, taking the case of A. in illustration, when we have located his lesion we have done anatomical work only; when we have noted all we can about his convulsion (therefrom inferring that the lesion is a "discharging lesion" and the effects of its discharge direct and consecutive), we have done physiological work only. The experimenters on animals do the anatomico-physiological work thoroughly well.

Taking up again the case of A., we have now the third element, pathology, to consider. Here is the great difficulty. Being sure that A. has a discharging lesion of this thumb centre, there may be no evidence to show whether that lesion is produced (indirectly) by tumour or not. Here I urge again the necessity of distinguishing between the physiology and pathology of cases of nervous disease.[42] Having urged that there is a degree or kind of functional change (*b*) which is not the result of any pathological process, I am all the more wishful to urge the distinction. I continue to speak of the first degree (*a*), and urge the distinction from this point.

It may be epigrammatically said, and with truth, that an epileptiform seizure is "only a symptom." Nevertheless, it is always symptomatic of one *physiological* thing, a "discharging lesion." Otherwise put, an epileptiform seizure is not a symptom of tumour, of "softening," or of meningitis; it is a symptom of the one thing—high instability of certain cortical cells, *however produced*—produced by any pathological process. Physiologically speaking, there is but one "cause" of epileptiform seizures, namely, high instability of some cells of some centre of the Rolandic region; but there are many "causes" of them if we mean pathological processes leading to that instability. So that in the case of A. (he being *subject to fits*), the question in pathology is not the vague one: "What is the *disease* of a certain part of his cortex"? but "What abnormality of the nutritive process has produced such an alteration in the composition of the material of cells in that part as to render them highly explosive, and how was that abnormality set up?" Or, more generally and regarding all cases of epileptiform seizures (and epileptic too), we put the question: "How are local persistent discharging lesions established and kept up?"

The first question is: "What is the most general nature of the abnormal nutritive process of cells of discharging lesions in epileptiform seizures?" This we ask ourselves, whatever the particular gross pathology or morbid anatomy, tumour, "softening," etc., may be in any case.

The cells of the discharging lesion, although *quasi*-parasitical, are not strictly parasitical; for another purpose I urged that the nervous arrangements into which they enter remain integral parts of the nervous system, and now I urge that they are nourished along with the rest of the body. Their nutrition does go on in some base fashion as certainly as that that of their stable neigh-

[42] I have urged this many times for at least twenty years. "Functional changes must not be confounded with pathological changes, although of course the two necessarily co-exist" ("Study of Convulsions," "St. And. Grad. Rep.," vol. iii, 1870). I mention this as I am said to have put forward the "theory of discharges" as the *pathology* of epilepsy—a thing I earnestly repudiate. In 1870 I did not make two degrees of functional changes, and spoke only of what I now call the first degree (*a*).

bours goes on in a proper way. By nutrition of some kind the cells of the discharging lesion attain high tension and very unstable equilibrium, and occasionally discharge suddenly, excessively, and temporarily; their stability is after their discharge below normal; by continuance of this abnormal nutrition they reattain high tension, or no more fitswould ensue. What is the "base fashion" of nutrition of these cells? It does not follow that the cells are *more* nourished, although they are certainly *worse* nourished. So to put it, they may be less nourished in quantity and worse nourished in quality. The nutrition must be such that it alters the composition of nervous matter of the cells, and in such a way that it becomes more explosive.

I have so often spoken on this subject that I will only mention the hypothesis that the nutrient fluid bathing the cells is comparatively stagnant, and that in consequence there is inferior nutrition. I suggest that there is "substitution nutrition," phosphorus compounds becoming more nitrogenous, or nitrogenous compounds more nitrogenised. I may illustrate by the well-known case of glycerine becoming explosive (nitroglycerine) when some of its hydrogen is replaced by nitric peroxide. The Composition of this substance is altered, but its Constitution remains the same. It may possibly be that the mass of the explosion of nerve cells, both in health and convulsion, is of non-nitrogenous matter, as is the case in the normal discharges of muscle. But the suggestion is that a nitrogenous substance is the "pivot" of the metabolism of nervous matter both in physiological and in hyperphysiological states.[43] I suppose the greater nitrogenisation of the material of nerve cells makes it highly explosive, but that the constitution of that material and the morphological structure of the cells remain the same. If this hypothesis be valid, there are presumably degrees of substitution (as there are in the three chloracetic acids, for example), and consequently degrees of high explosiveness. It may be that when a patient's fits "get worse," the original fulminate becomes more fulminant by still greater nitrogenisation, as well as that more cells become part of his fulminate. The abnormal nutrition leading to change of composition is a pathological process, whilst the high explosiveness it produces is a functional change. Surely it is the change of function which should be called a functional change. Whether these speculations be valid or not, it is a warrantable inference that the nutrition of the cells of the discharging lesion goes on, that it goes on wrongly, and that some material of the cells does become highly explosive.

Now I consider differences in sizes of cells with regard to their normal and morbid nutrition.

For theoretical reasons, and after the observations of Betz and Mierzejewsky, I suggested [44] that those centres of the motor region which especially represent small muscles (eyes, face, and hands) will have a greater number of small cells than those which especially represent the large muscles of the limbs. I think this is generally true of the "motor region." And I suggest that it accounts for the great frequency of onset of epileptiform fits in the hand and face, for reasons to be given presently. The "leg" centre contains many large cells, but it also contains some small cells. Bevan Lewis's researches, to which I am very greatly indebted, show that the parts of the motor region representing small

[43] "Whether the chief product of the metabolism of any tissue be a proteid substance, or a fat, or a carbohydrate, proteid substance is the pivot, so to speak, of the metabolism, and nitrogenous bodies always appear as the products of metabolism." (Foster, *Physiology*, pt. ii, p. 828).

[44] *Medical Press and Circular*, August 23rd, 1876.

muscles have most small cells. He says—and this bears on the remark I made on the leg centre—"that alongside the largest pyramidal cells are numbers of others of the *smallest* dimensions" (italics in original) [45] so that the discharging lesion of the "leg centre" may be made up of small cells.

Although it is convenient for some purposes to say "small muscles," the expression is not exact. For I urge once more that nervous centres do not represent muscles, but movements. I will substitute the expression "small movements" for "small muscles," and that of "large movements" for "large muscles." As these terms, the best I can think of, are vague, their meaning must be taken as defined here. Most of the movements of the hand are "small" according to the definition I now give of "small movements." The parts moved have little mass, and in most of the operations they serve in there is little more added mass. The muscles are small and numerous, and in most operations by the hand their movements are of little excursion, of short duration, and rapidly changing. Short, prompt, and frequent liberations of small quantities of energy will be required for these successions of "small" different movements; I submit that the nervous arrangements for these movements of the hand have small cells, and very many small cells. Most of the muscles of the shoulder are "large movements," according to the following definition. The muscles are large and few; there is much mass to move, the whole arm to lift, and the added mass is often great in some of the operations they serve in, as in lifting weights the hand takes up. In most operations they serve in, the movements are of large excursion, of comparatively long duration, and are comparatively little changing. They will require persisting and slow liberations of large quantities of energy by, I suggest, comparatively few and large cells. We may have "large" and "small" movements of different parts of one limb in a single operation. When the arm is put forth there are "large movements" of the shoulder, upper arm, and forearm; and when the fingers, thus put forward, elaborately explore an object, there are "small movements" of the hand.

But although parts which most often engage in "large movements" have large muscles, yet there may be "large movements" of parts having small muscles. If we grasp an oar and pull a boat, the whole of the musculature of the arm serves in a succession of similar large movements (and then subordinately to other movements). For these, I suppose, will be discharges of large cells, even for the movement of the hands grasping the oar; there is, indeed, then but one unchanging movement of the whole hand, a large movement *as defined*, all the small muscles serving together as if one muscle.

A principle of representation is here in question; we have not only to do with sizes of cells, but also with numbers of cells, and therefore with the volume of different centres. Schroeder van der Kolk, illustrating by the case of the sturgeon, which has large muscles and few cells in its spinal cord, pointed out that there is not a mere relation between quantity of grey matter and size of muscles, but that the grey matter is greater in proportion to the complexity of movements of muscles. This shows that we have never to forget that centres represent movements of muscles, not mere muscular masses. I have several times drawn attention to Herbert Spencer's statements bearing on this question.[46] One remark he makes is: "In proportion to the number, extensiveness, and complexity of the relations, simultaneous and successive,

[45] *A Text-book of Mental Disease*, p. 106.
[46] *Psychology*, 2nd ed., vol. i, pp. 35, 55, 67.

that are formed among different parts of the organism, will be the quantity of molecular action which the nerve-centres are capable of disengaging" Spencer takes count of both impressions and movements; I am illustrating by movements alone. The much greater volume of the middle motor centres is in accord with the fact that they represent vastly more numerous different movements than the lowest motor centres do; the muscles represented by both levels are, of course, the same, being in each case all the muscles of the body; hence the middle motor centres contain many more cells and fibres than the lower centres do. Presumably the same principle applies in detail. According to Horsley and Beevor, the thumb and index finger, which have a great number of different movements, chiefly "small movements," have a large area of representation in the motor region. As to the trunk area of the cortex, I quote what Horsley and Schäfer say:[47] "It certainly is not a little remarkable that the numerous and powerful muscles of the spine should be governed from so small a portion of the cerebral cortex, *but it is to be remembered that the movements of which the spine is capable are comparatively few and simple*" (no italics in original). The supposition is that parts having many small and greatly changing movements are especially represented by small cells and by many small cells, and that parts having but few and little changing ("tonic") movements are represented by large cells and by few large cells.

The size of cells is a very important matter with regard to their nutrition. Both in health and in disease small[48] nerve cells will be nourished more quickly than large ones when both are bathed in the same nutrient fluid. (From diminished nutrient supply the small cells will atrophy sooner than large ones.) The smaller cells will become highly unstable sooner than large ones during morbid nutrition. It certainly is the fact that most epileptiform seizures begin in parts having "small movements," in parts represented by areas of the cortex having most small cells. As I have pointed out, they may begin in the large muscles of the upper arm; it would be begging the question to say that in these cases the smallest cells of the "shoulder centre" are those which first become highly unstable. The size of cells bears also on rates of discharge.

Four hot iron balls will become cold much sooner than the same mass of iron in one ball having the same quantity of heat as the four balls together have. From discharge of four small cells, which are together equal in mass to that of one large cell, there will be, I submit, a liberation of energy in a shorter time than by the large cell, supposing equal quantities liberated in the two cases. Hence another reason for fulmination of the highly unstable cells of discharging lesions if these cells are small. As being somewhat illustrative, I may refer to different sizes of grains of powder used as ammunition; slowly burning (pebble)

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[47] *Phil. Trans.* vol. clxxix, 1888, B.

[48] "Other things equal, the smallest cells will soonest become unstable. A mass of nervous matter in many small cells will 'present a much larger surface to the contact' of nutrient fluid than the same mass in a few large cells" (Harveian Lectures, *Med. Times and Gaz.*, January 11th, 1879). My attention was first directed to this subject on reading Spencer's *Biology*, where he expounds his theory of growth. A brief statement of Spencer's theory will be found on p. 220 of *The Evolution of Sex*, by Geddes and Thomson, from which I quote "In spherical and all other regular units the mass increases as the cube of the diameter, the surface only as the square." I refer the reader to Ross's great work, *Diseases of the Nervous System*, vol. i, p. 13, where he deals with the significance of differences in sizes of nerve cells with regard to normal and abnormal nutrition and its consequences.

powder is required for large cannons, quickly burning (fine-grained) powder for firearms.

To repeat the several hypotheses, Epileptiform seizures begin most often in parts of the body having "small movements;" these movements are represented by nervous arrangements having many small cells.[49] Small cells present a more extensive surface to nutrient fluid than the small quantity of grey matter in large cells, and will be more quickly nourished than large ones are. Nerve cells become highly unstable from an abnormal nutrition, such that, although their structure and the constitution of their material is unaltered, that material becomes of more nitrogenous composition, and thus more explosive. Small cells become highly unstable more readily than large ones do; thus discharging lesions are supposed to be especially of small cells. A rapid liberation of energy overcomes greater and more numerous resistances than a slower liberation of an equal quantity of energy. Small cells liberate their energy in a shorter time than large ones; hence the currents developed by fulminates of small cells overcome greater and thus more numerous resistances than would fulminates of large cells, and hence produce more convulsion and greater range of convulsion.

We have assumed that the nutrition of the cells of the discharging lesion is continuous, and have supposed in effect that the nutrient fluid is comparatively stagnant. But how is this comparative stagnation brought about? This brings us to pathology, commonly so-called. We have to distinguish between what I may call the coarse pathology of a case and its immediate pathology.

[In the remainder of the lecture the production of discharging lesions by tumours and by arterial occlusion was considered.][50]

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[49] The movements, speaking most generally, represented by the cerebrum are, I suppose, numerous different punctuated movements (many and different "small movements"). Presumably those represented by the cerebellum are, in comparison, few and little different movements, movements as it were gliding into one another (few and similar "large movements"). It is interesting to observe that the structure of the cerebellum is more uniform than that of the cerebrum, and that those of the cells of the cerebellum which are presumably motor are large and of nearly equal size. The movements for bracing up the spine in standing or walking, and the separate movements of the legs and arms in walking, will require comparatively continuous supplies of large quantities of energy. (I believe, however, that the cerebrum and cerebellum are both engaged by contemperation in at least all extensive operations.)

[50] See JOURNAL, July 21st, 1888.

Lecture III.

After-Effects of Epileptiform Fits.—Paralysis due to Cortical Exhaustion.—Analysis of a Case.—Variations in Degree and in Range of Post-epileptiform Paralyses.—Hemiplegia, Post-epileptiform and from Destruction of Motor Part of Internal Capsule contrasted.—Post-epileptiform Defects of Speech.—The Negative and Positive Elements of these Defects.—The Psychical and Physical.—The Concern of the Right and of the Left Half of the Brain in Speech.

I HAVE now to consider the After Effects of excessive nervous discharges in cases of epileptiform seizures. This part of our subject is of extreme importance; the presumption is that from the study of the comparatively simple post-epileptiform conditions we shall obtain a basis for the interpretation of post-epileptic conditions. It is for this reason that I go into some detail as to the former.

There is not rarely after epileptiform seizures local temporary paralysis;[51] and sometimes aphasia with it. For the present I speak of paralysis only. In this inquiry we have no direct concern with paralysis, temporary or permanent, owing to destruction (by clot, softening, or any other pathological process) of any part of the brain, not even if the malady set in with a convulsion.

I must particularly mention cases in which there are both (1) permanent paralysis—for example, imperfect hemiplegia; and (2) occasional epileptiform seizures, the convulsion beginning in some point of the muscular region paralysed and involving part or the whole of it, and sometimes also parts beyond that region. In these cases the two opposite functional states—destruction of some nervous elements which represented complex movements, and high instability of some others which still represent other complex movements of the same muscles—are side by side in one part of the cortex (or the latter of cortex and the former of fibres passing down from it). After a paroxysm in such a case there may be temporary increase of the paralysis. For my present purpose, which is only an attempt to explain the nature of the sequence (paralysis after convulsion), I shall exclude these cases, and deal only with what I may call uncomplicated cases—that is, with those in which there is only paralysis immediately when epileptiform seizures have ceased, such paralysis being temporary (unless, of course, the fits recur at short intervals). Something has to be said, however, regarding certain of the so-called uncomplicated cases.

In some cases in which there is no *discoverable* paralysis in the intervals of the fits, except for a short time after one, there may be indirect evidence of a negative functional state of a few fibres of the second segment of the kinetic route. That evidence is foot clonus and exaggerated knee-jerk on the same side as that in which the convulsion occasionally starts. I mean, of course, cases in which these abnormal tendon reactions are producible at all times, although in a greater degree just after a fit. It may be that severely careful tests would show some *quasi*-trifling paralysis

[51] This paralysis was long ago described by Todd under the name "Epileptic Hemiplegia."

—loss of a very few complex movements. I shall here ignore this slight inferential paralysis, and speak of such cases as "uncomplicated."

The term post-epileptiform paralysis will be used to include all paralyses the immediate sequels of epileptiform seizures. Arbitrary divisions may be made, speaking of range only. (1) Terminal paralysis, as of a hand; (2) monoplegia, as of an arm; (3) hemiplegia; and (4) a range which is not generally admitted—some degree of slight universal paralysis. I hold the hypothesis (essentially that of Todd and Alexander Robertson) that there is exhaustion of central nervous elements, including fibres of the second segment of the kinetic route, and that this is produced by the sudden and excessive discharge in the prior paroxysms These nervous elements are exhausted, not otherwise injured, so that recuperation is prompt, and the corresponding paralysis is temporary. There is a sequence of two opposite functional states, superpositive of the first degree (*a*) in the paroxysm, and then, after the paroxysm, negative. But there is often more than a negative functional state in the post-epileptiform condition. We may find increased tendon reactions implying a superfunctional state of the second degree (*b*) of lowest motor centres. The duplex nature of these post-epileptiform conditions must be borne well in mind.

The term "exhaustion" has been objected to; general bodily prostration is, of course, not meant; but, as said, exhaustion of nervous elements in a particular part of the central kinetic route. There are, I suppose, degrees of exhaustion, and no doubt of different numbers of nervous elements of the kinetic route in different cases. But illustrating by the extremest degree, the supposition is that nervous elements of the route, after their excessive "exercise" in the fit, are left "fatigued" to the degree of utter impotence. The nervous elements exhausted in post-epileptiform paralysis are, I suppose, in the same state as are the motor nerve fibres of the sciatic nerve going to the cut-off leg of a frog after strong faradisation of that nerve trunk; the leg is convulsed by the faradisation (stage analogous to epileptiform seizure), and is next paralysed (stage analogous to post-epileptiform paralysis) because its motor fibres are exhausted by the unnaturally high functioning they have been artifically compelled into. A closer analogy may be stated, although the case to be instanced is a complicated one. The motor nerves of a frog, poisoned by a very large dose of strychnine, lose function partly by the direct action upon them of that poison, but partly (this is what is relevant) through exhaustion—"the exhaustion of over-use due to the intense activity of the nerve during the stage of spasm."[52]

Not only the term, but the hypothesis itself is objected to. I will consider other hypotheses. The paralysis has been ascribed to cerebral congestion consequent on arrest of respiration in the preceding seizure. It is difficult to see how cerebral congestion, even if we grant that it can produce paralysis, could be so exquisitely local as to produce what we sometimes observe, paralysis of one arm only, or even of but part of one. Again, absolute paralysis of a limb is found after seizures nearly limited to that limb, in which seizures there was no arrest of respiration and (if that is thought to bear on the question) no loss nor even defect of consciousness; the patient may talk throughout an attack in which an arm is involved, and after which it is temporarily paralysed. There is the hypothesis that the temporary post-epileptiform paralysis is owing to a small extravasation of blood in the brain,

[52] See Wood's *Therapeutics*, 7th edition, pp. 258-9.

caused, I suppose it is meant, by congestion consequent on arrested respiration in the preceding seizure. I admit that very small clots may cause local paralysis, and that such paralysis may be transitory. But the post-epileptiform paralysis is—that is in my experience—always of the parts which were first and most convulsed in the prior fit; it would be marvellous if a small hæmorrhage happened locally in one-half of the brain, so as to produce temporary paralysis of the parts first and most convulsed, and happened in the same place in every fit the patient had. Besides, if arrested respiration could cause cerebral hæmorrhage by leading to sudden great congestion of the brain, we should find very local paralysis after fits of the epileptic kind, and we do not; according to current opinion there is no paralysis at all after seizures of this kind. I shall exclude the two hypotheses just dealt with.

Post-epileptiform paralysis has been ascribed to inhibition by some medical men whose hypotheses deserve respectful consideration. Gowers believes that discharges in epileptic fits sometimes inhibit; he thinks that temporary paralysis is found in some cases after a purely sensory discharge which does not next discharge motor centres, but inhibits them. In some cases of epileptiform seizures the patient tells us that his arm "falls dead," there being no spasm in it, whilst the face of the same side is being convulsed. This, so far as I can learn, is a paroxysmal, not a post-paroxysmal paralysis, but possibly it remains for a short time after the convulsion of the face has ceased. I express no decided opinion as to the validity of the inhibition hypotheses. I think it possible that there may be discharge spreading slowly in a motor centre of the middle level, excessive enough to cause slight after-exhaustion of some of its elements, although not one strong enough to overcome the resistance of lowest motor centres, and thereby to produce actual convulsion. I have spoken of "feelings of convulsion" in which there is no actual convulsion, but do not know whether there was any degree of post-paroxysmal paralysis of the parts "ideally convulsed." I go on to consider how far the hypothesis I hold, essentially that of Todd and Robertson, accounts for the facts of different cases.

There is exhaustion of the cortex after artificially induced epileptiform fits in some lower animals, as Franck and Pitres[53] have shown; they use the term "épuisement cortical post-épileptique." They write:[54] "Le phénomène de l'épuisement cortical consécutif aux accès d'épilepsie partielle (what I call epileptiform seizures) est très facile à constater." The "épuisement" is transitory; it lasts a quarter or half an hour. By artificial excitation of the part of the cortex in question, as the exhaustion diminishes, simple movements are first producible, and later epileptiform seizures, but not, for some time, is an attack provoked so intense as before the "épuisement." I think that the researches of these distinguished physicians countenance the inference I have drawn that the exhaustion in post-epileptiform conditions, although local, is yet widespread in the "motor region;" that besides "running down" of the cells of the fulminate there is also running down of the collateral normal (stable) cells which were compelled by the fulminate to discharge excessively. Franck writes:[55] "Cet épuisement est tout *local*, non point qu'il se borne exclusivement à la portion circonscrite du champ moteur excité en premier lieu, mais il est limité à la zone motrice de ce côté." Exhaustion in the sense of general bodily prostration is not meant, for Franck and Pitres point out

[53] *Arch. de Phys.*, 1883.
[54] *Op. cit.*
[55] *Fonctions Motrices du Cerveau*, pp. 90, 91.

that excitation, without effect on the exhausted part of the motor region, when transferred to the motor region of the opposite hemisphere, produces severe convulsions.

I will here mention an objection to the hypothesis of exhaustion made to me by a physician whose opinion I respect highly, that during post-epileptic coma there may occur a convulsion affecting all parts of the body, which I say are then paralysed. But what I mean by paralysis in post-epileptic coma is loss of some most complex movements, represented by the highest centres of all or very many parts of both sides of the body; there is retention of other most complex movements represented by those centres; most of the complex (middle centres) and simplest movements (lowest centres) being also retained. Further, it is quite certain that an arm which the patient is quite unable to move in the slightest degree after an epileptiform seizure may be suddenly seized with another severe convulsion (I am supposing a case in which neither in nor after the paroxysm consciousness is lost). This, however, is only reasoning by analogy, and I admit that the interpretation of the last-mentioned case is to me most difficult.

I will now give an illustration of post-epileptiform paralysis. It may be taken to be an artificial one, but it is essentially that of a patient whose case I reported.[56]

A man, B., was *seemingly* (1) quite well when he arrived at my house after a walk of about a mile. (It is convenient to speak of this as the "first stage," not, of course, of the fit, but of the dramatic occurrence I am relating.) (2) (*a*) A fit began in the toes of his left foot.[57] (*b*) The whole leg was gradually involved, the spasm passing up the limb. In about eight or ten minutes the convulsion ceased, when (3) the leg was found to be (*a*) paralysed, and (*b*) there was exaggeration of its knee-jerk and clonus of the foot. (4) In six hours or less he was *seemingly* well again, certainly he was then rid of his paralysis; and next day (I did not test his tendon reactions before) his knee-jerks were normal and there was no foot-clonus. Let me consider the four acts of this drama.

(1) The patient was not really well when he arrived at my house. He had a persistent discharging lesion, presumably of a few cells of his leg centre (perhaps only of some of those of the hallux centre); so to say, he always carried it about with him, or, to speak more precisely, it was a persistent *quasi*-parasitical hyper-functionable part of himself.

(2) (*a*) The discharging lesion did function and that excessively, and possibly produced the initial spasm (of the toes) by its sole discharge; but (*b*) next, as a fulminate, by overcoming the resistance of (discharging) other cells of the leg centre it compelled these normal stable cells to discharge—compelled them to co-operate in its excess.[58] So much for the discharges (primary and secundo-primary)

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[56] *Medical Times and Gazette*, February 12th, 1881.

[57] Referring to the real case (*op. cit.*) the patient said that in all his seizures the first "sensation" was to the outer side of the ball of the great toe; very likely I missed a very earliest stage, one possibly of spasm limited to the big toe.

[58] In the real case (*op. cit.*) the left arm was slightly convulsed. The current hypothesis would be that, in causing this part of the fit, the discharge spread from the leg centre to the arm centre. This I cannot disprove. Yet I think it an equally legitimate hypothesis that the discharge causing the slight movements of the arm in this case was of those elements of the "leg centre" representing subordinate movements of the arm. No doubt, of course, in a severer fit other centres of the motor region would be discharged. As implied in several parts of these Lectures, I do not accept the current doctrine of Localisation. The minute investigations of the monkey's cortex by Horsley and Beever go strongly against it. To me the "leg centre" is only a part of the "motor region" where most special movements of the leg are represented, and where

of the cortex (middle motor centres). That there was a super-normal activity from cortex excessively discharging to and of the muscles of the leg convulsed is certain. The route of the numerous and rapidly succeeding nerve impulses from the hyper-physiological cortical process would be certain fibres of the corona radiata, of the internal capsule, of the crus cerebri, pons, and medulla of the right side, then of fibres of the left lateral column (possibly also of the second and third sets of interconnecting fibres, both of which I shall ignore here) up to the anterior horns (some lowest motor centres); so far the second segment of the kinetic route is concerned. The resistance of the cells of these lowest motor centres would be overcome (secondary discharges), and the impulses (presumably increased in number) would then be of fibres of the nerve roots from those centres, thence of the continuing fibres of nerve trunks and their branches to the end plates of the muscles (third segment of the kinetic route); the resistance of the muscles would be overcome, and there would finally be great "explosive decomposition" of muscle substance (tertiary discharges).

(3) The paralysis signified exhaustion of nervous elements previously excessively functioning in the paroxysm. There would be exhaustion of cells of the cortex, not only of those of the discharging lesion, but also of those collateral stable cells which it, as a fulminate, compelled to discharge excessively. There would be exhaustion, too, of the fibres passing down from both sets of cells. The hyper-kinetic route, or at least part of it, would now be a hypo-kinetic tract. There would be exhaustion as low down as certain lowest motor centres; these centres were therefore exalted in function (second degree of superpositive functional change), hence the increased tendon reactions.

(4) The paralysis had passed off; the patient could walk well. Next day the knee-jerks were normal. There was recuperation of the exhausted nervous elements. No doubt the cells of the discharging lesion (after the fit, stable below normal) began at once by gradual morbid nutrition to re-attain high tension and very unstable equilibrium—"began to prepare for the next fit."

So far as I have seen, post-epileptiform paralysis is always most of the parts which were first and most convulsed in the preceding paroxysm, as in the case of the patient B. This correspondence is denied by some eminent physicians; further observations will settle the matter. It is an important one, for, according to the view I take, post-epileptiform paralysis adds no evidence to that afforded by the prior convulsion as to the seat of pathological changes in the cortex. If, however, there is not the correspondence I suppose, the case is very different and the situation is a most difficult one.

I think there is a relation of proportionality between the severity of the epileptiform fit (severity of the discharge) and the subsequent temporary paralysis (amount of exhaustion). I should have thought this a truism if it had not been expressly denied. It is averred that whilst after the epileptiform kind of

subordinate movements of the arm and of other parts of the body are also represented (the same, *mutatis mutandis*, for other centres). The researches of Sherrington (*Journ. of Physiology*, vol. x, No. 5 seem to me to be counter to the current doctrine. He writes, ".....after lesion in the leg area, encroaching little if at all upon the cortex of the arm area, the degeneration in the cord stopped short in great part in the cervical region, especially in the cervical enlargement. Again, after lesion in the arm area, encroaching little if at all upon the cortex of the leg area, the degeneration in the cord extended down through the dorsal into the lumbar and even throughout the sacral region of the cord." He suggests, however, that the fibres descending from the arm area into the lumbar region are visceral.

seizure there is often decided paralysis, there is not, as a matter of fact, any whatever after the severer fits of the epileptic kind. I demur to the "fact," believing that whilst after a limited epileptiform seizure there is often great *local* paralysis, loss of *many movements* of a small part of one side of the body, there is after a severe epileptic fit *widespread* paralysis, loss of *few movements* of most, if not of all, parts of both sides of the body. Indeed, I submit that the whole condition of bodily impotence after a severe epileptic fit is paralysis, and that, speaking generally, there is really more paralysis than is found after severe epileptiform seizures.

The relation of an epileptiform fit to the sequent paralysis, or, more exactly, the relation of the two opposite functional states, discharge, and subsequent exhaustion, is not a simple relation.

According to the hypothesis I hold, there should always be some, however little, paralysis after an epileptiform seizure, at least some in the parts first and most convulsed, however slight the attack may have been. Trifling degrees of paralysis are easily overlooked. This deserves remark. Once more I urge that more or less paralysis from negative central lesions is always loss of more or fewer *movements;* it is not to be thought of merely as *loss of power in muscles.* There may, for example, be loss of the most special (the most "delicate") movements of the muscles of the hand with retention of other, the next most special (the "coarser") movements of those muscles. I will illustrate by a case of imperfect post-epileptiform paralysis.

After a slight epileptiform seizure starting in the hand, the patient may have difficulty in picking up a pin, and may yet be able to grasp strongly. There is clumsiness of movement, or perhaps some may say "loss of muscular sense." These terms must not let us overlook the fact that the slightly abnormal motor condition of the hand is a double condition, one of two opposite elements. There is in the case supposed difficulty in picking up the pin, because a few most special movements of the hand are lost (so far paralysis, negative element); the pin has to be picked up by the next most special movements remaining, which are *now* the most special of the movements (positive element); they do not serve so well as the most special would have done; hence the operation is "clumsy."[59]

From such *quasi*-trifling paralysis as that indicated, loss of a few most special movements of the muscles of the hand, there are met with after severer epileptiform seizures beginning in the hand degrees of loss of next and next most special (or, equivalently, of the next and next more general) to loss of all movements of the muscles of it, and of some movements of the rest of the arm too; there is then no "clumsiness," for no movements of the hand remain to do anything.

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[59] Here is an illustration of the principle of duplex symptomatology of nervous maladies, and that the positive element is often owing to activity or to overactivity of nervous arrangements untouched by any pathological process. The movements by which the pin is picked up are the outcome of activity of healthy nervous arrangements. To blame these, to speak figuratively, for the clumsiness of the operation is to ignore that they are doing their best in the evil circumstances. The principle is most important, if it applies, as I think it does, throughout the nervous system, from the symptomatology of a case of paralysis of an ocular muscle to that of cases of insanity; in the former case it is quite certain that most of the symptomatology answers to activity of perfectly healthy nervous arrangements of the highest centres: the negative mental symptoms in cases of insanity alone correspond to disease; they signify Dissolution of part of the highest centres; the coexisting positive mental symptoms signify Evolution going on on a lower level of those centres. The very same principle is displayed in cases of aphasia.

The last remark introduces another part of our subject. It implies that post-epileptiform paralysis varies in a double way, (*a*) in degree (number of movements lost) and (*b*) in range (number of parts of which movements are lost). I think, in harmony with what was said in Lecture II, on compound order of spreading of convulsion, that degrees of difference between what may be called little and great paralysis after epileptiform seizures are not simple degrees of more or less amount of paralysis, nor simple degrees of more or less range; there are degrees of both, there are differences of Compound Degree. After the slight epileptiform seizure alluded to in the foregoing, there was slight paralysis limited to the hand. If that patient had had a somewhat severer fit, he would have had after it more paralysis of the hand (loss of more movements of it) and greater range of paralysis (loss of some movements of the next part, say the forearm).

That there was exhaustion lower than the cortex in B.'s case, exhaustion also of fibres of the second segment of the kinetic route, that is, up to the lowest motor centres, is signified by the exaggerated tendon reactions. I have spoken of the lowest motor centres and of the third segment of the kinetic route (fibres from them to the muscles) and of the muscles as not being exhausted in B. at the time when his leg was paralysed. It would, however, be remarkable if a few elements of these lowest parts of the kinetic route were not hypo-kinetic to some trifling degree after the hyper-kinesis during the convulsion—if there were not after their excessive exercise some exhaustion of a few of their elements. No doubt, being more strongly organised structures, the lowest motor centres and muscles are less easily exhausted by the discharge, and, if exhausted, recuperate soonest of all parts of the route. After some epileptic fits (Westphal, Gowers, Beevor) the knee-jerks are temporarily lost; in these cases some lowest motor centres, the lumbar nuclei (as Gowers suggests) are probably temporarily exhausted. For my part, I think it likely that during the post-epileptiform paralysis in B., although his knee-jerks were exaggerated, there was some trifling hypo-kinesis of the lowest part of the route, that the hypo-kinetic tract was from cortex up to and of the muscles, although, no doubt, elements of the cortex and of the first segment of the kinetic route were most exhausted. The muscles in such cases act to faradisation (I did not test B. this way) as well as do those of the fellow leg. But if we were to take a case of paralysis, such as that of a man a few years ago under my care who had nineteen hundred and forty-five fits—each beginning in his left thumb, involving the left arm, fixing his chest, and turning his head to the left—in about fourteen days, and whose left arm was absolutely paralysed in the intervals, we might find by fine testing (what we did not find in that patient by ordinary testing) some slight degree of reduction of faradic reaction of the muscles, or, if not, of reduction of faradic or galvanic reaction in their nerve-trunks, of the part paralysed. In B. the exaggeration of the knee-jerk would not show that *all* the cells of the anterior horn which is concerned with that jerk were intact; a few of the smallest cells may have been exhausted.

In a case of amyotrophic lateral sclerosis there is wasting of some cells of anterior horns (hence the atrophy of some muscular elements) and increased activity of other cells of the same horns (hence the increased tendon reactions of the same muscles). This case shows that the functional condition of different cells of the same anterior horns may be diametrically opposite, and that the symptomatology in the muscular region which the horns supply may be of two correspondingly opposite elements. I shall put

aside the hypothesis of exhaustion of a very few elements of the lowest motor centres, and of the third segment of the kinetic route, and of the muscles in such cases as that of B. for the present, but yet do not abandon it. I shall speak of the hypo-kinetic tract in such cases as that of B., as if it extended no lower than fibres of the lateral column, and as not involving any cells of the lowest motor centres in which they end. It would be reasonable to grant that it was at least of that depth in B.'s case.

It will be well to draw attention to resemblances and differences between post-epileptiform hemiplegia and the hemiplegia which is the result of destruction of the motor part of the internal capsule; for the moment assuming that the explanation given of the former is correct, and that the statements accepted by most physicians as to the latter are accurate. I will call the former Hemiplegia E, the latter Hemiplegia D.

First, for resemblances, taking to begin with, for comparison and contrast, *recent* Hemiplegia D. The regional (paralytic) affection is the same as in Hemiplegia E, and in each depends on a negative state of the same strand of fibres, those interconnecting the right middle motor centres and the left lowest motor centres. (I shall speak only of what I called the first set of fibres.) Now for differences. In Hemiplegia E (as in the case of the patient B.) there is exaggeration of the tendon reactions, in Hemiplegia D they are, we shall suppose, normal. Correspondingly for the negative lesions; in Hemiplegia E the negative lesion is of the whole length of the interconnecting fibres mentioned (and spoken of before as the second segment of the hyper-kinetic route become a hypo-kinetic tract) up to, but not involving, the lowest motor centres. In recent Hemiplegia D the negative lesion is quite local, a breaking up of the capsular part of the interconnecting fibres; the rest of the segment, its whole extent below the capsular break, being normal,

Now for *old* cases of Hemiplegia D; in these there are increased tendon reactions; in them a negative lesion secondary to the capsular one has been established; there is then a negative state (destruction) of the fibres (lateral sclerosis) below, and added on to, the local capsular lesion;[60] that is to say, in the case of Hemiplegia E there is established at once the state of things which only occurs late in Hemiplegia D. So long as the exhaustion lasts (so long as the central kinetic route is hypo-kinetic) in Hemiplegia E, the situation is the same as in old cases of Hemiplegia D; it is the same for the time being whether the nervous elements in question are functionally dead, as in the former, or actually dead (broken up and wasted away), as in the latter.

In a popular use of the word cause, these negative conditions may be said to cause the increased tendon reactions, just as, popularly speaking, opening floodgates is said to cause water to flow. But using the word cause properly, it is an impossibility that the negative functional state in either Hemiplegia E or D can cause anything; a nothing cannot cause the something, the superposive condition for exaggeration of the knee-jerks. If Bastian be right in his conclusions as to the effect of absolutely complete transverse lesion of the dorsal or cervical cord (see Lecture I), the hypothesis of a change in the anterior horns, produced by any sort of pathological process, is no longer tenable, as he has pointed out. I shall, however, consider other arguments against that hypothesis, which are supplied by cases of post-epileptiform paralysis.

It is agreed upon that the increased tendon reactions in cases

[60] Sherrington has traced the "descending wasting" in the lateral column of the cord of the monkey as low as the coccygeal nerve roots.

of hemiplegia depend on exalted "excitability" (a superpositive functional state) of nervous elements of some anterior horns. There are different hypotheses as to the process by which that exaltation is produced, and thus as to the exact state of the cells of the anterior horns concerned. Two questions may be asked. Is the abnormal condition of the cells the result of any pathological process involving the horns themselves, or are the cells healthy although in greater activity? I reply to the first question negatively, and to the second affirmatively.

I take, for further consideration, an old case of Hemiplegia D, one in which lateral sclerosis is established. And now I consider rigidity as well as increase of the knee-jerk (limiting illustration to that one tendon reaction), both of which superpositive phenomena it is agreed upon imply exaltation of function of cells of anterior horns. One hypothesis as to the production of exaltation of function of the anterior horns (some of the lowest motor centres) is that the same process which leads to destruction of fibres of the lateral column produces, when come to the horns with which they are in physiological union, increased "excitability" of their cells; the same process, on this hypothesis, produces loss of function (destruction) of fibres and its physiological opposite superfunction of cells. The hypothesis of Charcot deserves to be received with profound respect. Referring to the condition of the cells of the anterior horns, as it corresponds to rigidity in hemiplegia, he considers it the theory which best accounts for facts we daily witness in cerebro-spinal diseases, that there is a purely dynamic lesion of their ganglion cells—a state analogous to that produced by strychnine. If this state of the cells be produced by an extension to them of the same process as that which has destroyed fibres, it (however slight the alteration effected may be, and however insusceptible of microscopical demonstration the change may be) must be considered to be the result of a pathological process.

This hypothesis cannot apply to Hemiplegia E; in this case it is not likely that there are caused by the excessive discharge a negative state of fibres, and a super-positive one of cells. All the more that after some epileptic fits there is found exaltation of the knee-jerk, and after others loss of it; differences, explainable on differences, in the severity of the discharge in the paroxysm, and on the different "depths" of exhaustion after it; there is a negative functional state in both, but lower down in the kinetic route, of cells of anterior horns, when the knee-jerks are absent.

My hypothesis is that both in Hemiplegia E and in *old* cases of Hemiplegia D, the cells of the anterior horns are perfectly healthy. They are, I submit, untouched by the pathological process causing the lateral sclerosis or by any other, and are simply over-active, partly from loss of control, and partly from non-antagonised cerebellar influx (second degree (*b*) of superpositive functional change). But I do not state the hypothesis of loss of control as I used to do. There is sometimes exaggeration of the knee-jerk in recent cases of hemiplegia, and this may be owing to sudden loss of cerebral (middle motor centres) control. I shall, however, speak as if the current opinions were correct—that both the increased tendon-reactions and rigidity "wait for" the descending degeneration in cases of Hemiplegia D. I now adopt an hypothesis put forward by Gowers, which is to the effect that close upon the anterior horns there are small inhibitory centres, the anterior horns being, in his nomenclature, "muscle-centres."

In cases of recent Hemiplegia D, before the degeneration in the ateral column, these local inhibitory centres are intact, and the

knee-jerks are not exaggerated. But I suggest that in such cases of Hemiplegia E as that of the patient B., they are exhausted as well as are fibres of the lateral column; after those epileptic fits, when the knee-jerks are lost, both the inhibitory centres and the muscle centres are exhausted. Now taking old cases of Hemiplegia D, and dealing with rigidity as well as with the exaggerated knee jerk, I would say that the "descending" process, when it reaches the anterior horns, destroys the small local inhibitory centres; the anterior horn proper, "the muscle centre" now bereft of its inhibitory centre, is uncontrolled. The cells of the "muscle centres" are not invaded by any pathological process; they are healthy, but yet over active from loss of control This hypothetis as to old cases of Hemiplegia D invokes but one mode of action—destruction. That hypothesis brings the facts of Hemiplegia E into harmony with those of long-standing cases of Hemiplegia D; it accounts for the absence of increased tendon reactions in recent cases of Hemiplegia D. Certainly cases of Hemiplegia E have to be considered as well as cases of recent and old Hemiplegia D in all hypotheses as to the nature of increase of the knee-jerk in cases of brain disease.

Of any degree of rigidity of muscular regions paralysed—during the paralytic stage or part of it I mean of course—after epileptiform seizures I know nothing. Possibly I have overlooked it. But Franck and Pitres have made very important observations on a muscular rigidity after epileptiform seizures produced in dogs and cats. Of course it might be said that this active muscular condition is owing to a slighter degree of the same cortical discharge which produced the obvious fit—that it is not post-paroxysmal, but a great attenuation of the paroxysm itself. But these distinguished observers tell us that the rigidity is altogether different from the convulsion in the attack itself. And what is decisive is that ablation of the whole motor zone does not cause disappearance of the rigidity.[61] It seems to me that the hypothesis I have taken from Gowers, exhaustion of inhibitory centres, and consequent over-activity of the muscle-centres of the anterior horns to which they belong, best explains these phenomena.

Although beyond mentioning it I have not in this Lecture spoken of cerebellar influx, I believe it to be a factor in the causation of the exaggerated tendon reactions in Hemiplegia E, and of these and the rigidity in old cases of Hemiplegia D—that in both there is loss of control and unantagonised cerebellar influx, and thus that the two positive symptoms are owing to the unhindered activity of perfectly healthy structures.

In some cases of epileptiform seizures at, or close upon their onset, there is loss or, at least, defect of speech with very little local spasm. (We have, of course, no concern in this inquiry with any cases of *le petit mal*). I shall, however, speak only of temporary abnormal affections of speech, not in, but after the paroxysms—of what is present when all convulsion is over.[62] I explain post-epileptiform defects of speech in the same way as I did post-epileptiform paralyses, supposing there to be in the former exhaustion of nervous arrangements of "Broca's region."

Just as the e are cases of epileptiform seizures with permanent paralysis of the side of the body in some part of which the convulsion starts and in which there is more paralysis, temporarily,

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61 *Les Fonctions Motrices du Cerveau* by François Franck pp. 88, 89.

62 "There is a peculiar class of cases of epileptic hemiplegia in which the exciting cause of the epileptic fit at the same time damages or greatly injures voluntary power and speech." (Todd, *Nervous Diseases*, lecture xv.)

of that side after a paroxysm, so in some cases of epileptiform seizures there may be permanent defect of speech and a temporary increase of that defect after a paroxysm. I shall omit consideration of these important cases. So far as I have seen, abnormal affections of speech are found after fits with right-side "signal symptoms." They certainly occur after left-sided fits, as Dr. Herman Weber has pointed out.[63]

I have only once known abnormal affection of speech to occur after a fit beginning in the foot (right); that case is of no particular value in localisation, as at the necropsy two large blood cysts were found, causing extensive compression of each cerebral hemisphere. Temporary defect of speech after epileptiform seizures beginning in the (1) right hand or (2) right side of face or tongue, or both, is not rare.

The "abnormal talking" after some eleptiform seizures has long seemed to me[64] not to be what could commonly be called aphasia; it often sounds like a *mélange* of an articulatory defect and of a speech defect. It may be that in these cases there is not only exhaustion of some elements of Broca's region, but also of elements of the corresponding lowest (bulbar) motor centres; if so, the situation is analogous to cases where, after epileptic fits, the knee-jerks are absent, from, presumably, exhaustion of (lumbar) lowest motor centres. I think the "abnormal talking" referred to most likely to occur after epileptiform fits beginning in the cheek or tongue. What may be distinguished as "pure aphasia" is itself, physically regarded, a paralytic affection in the sense of loss of complex ("articulatory") movements of the tongue, palate, lips, etc.; so the *mélange* spoken of, if the hypothesis suggested be valid, is physically a mixture of loss of some complex (middle level) and some simplest (lowest level) movements of those parts. The subject is not a simple one, because along with right hemiplegia from destructive lesions we sometimes meet with a difficulty of articulation (I used to call it "ataxy of articulation") when there is no obvious disability in the tongue, palate, lips, etc.[65] There is one thing which must be mentioned in this connection. We should not consider that the inability of a patient who is more or less aphasic after an epileptiform seizure to put out his tongue when told is evidence of implication of lowest motor (bulbar) centres; for in these cases the tongue moves well in other and simpler operations. Moreover, this inability is often found in cases of aphasia from destructive cerebral lesions, and lasts too long in those cases for the easy explanation of "shock;" in them implication of bulbar centres is out of the question. This curious symptom is loss of a most special movement from a cortical or sub-cortical lesion; in that sense it is paralysis. It is no way regarded a speech or articulatory defect. To put out the tongue when told is what is called a "voluntary" movement, just as lifting the arm when told, or the card-sharper's shrug (Lecture II) is.

I shall ignore the hypothesis mentioned, and deal only with cases which would be admitted by all to be defects of speech proper, that is to be "of an aphasic character"—cases in which much speech of an imperfect kind remains, the utterances being

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[63] *Trans.* International Med. Congress, 1881, vol. ii, p. 19.

[64] "Study of Convulsions," *St. And Med. Grad. Assoc.*, vol. iii, 1870; Reynolds's *System of Medicine*, vol. ii, second edition, p. 287; see also Gowers' *Epilepsy*, p. 101.

[65] Dr. Charles K. Mills, of Philadelphia, relates, *University Medical Medicine*, November, 1889, a very important case of "Softening of the Face-area with Oro-lingual Monoplegia."

clearly articulated. In most of these the prior convulsion starts in the right hand, and, I have thought, most often, not in the thumb or index finger, but in the hand generally or in the ulnar fingers. Of course there are many degrees of abnormal affection of speech of this kind, and it varies according to the time elapsed since the cessation of the fit.

I have not observed post-epileptiform *loss* of speech, but only defect of speech ("partial aphasia"). I shall use the term Defect of Speech E, or Partial Aphasia E. The term "defect of speech" is equivocal, as is also the term "partial aphasia;" it really covers two opposite elements, negatively loss of some speech, and positively retention of the rest of speech.[66] The patient gets words out clearly, and may even get out simple appropriate replies, such as "Very well," and may answer correctly by "Yes" or "No," or both. This is the positive element: it is the inferior speech to which the patient is reduced. But he is *not* able to reply correctly except by "Yes" and "No," and by other simple and very general expressions; he is *not* able to converse properly on simple subjects; he is *unable* to explain anything at all complex. These statements give the negative element: the speech lost. It is of extreme importance to distinguish the two opposite elements of the symptomatic condition. It is plainly impossible that the post-epileptiform exhaustion (*vide supra*), which I suppose there is of part of Broca's region, can answer to the patient's utterances—to the positive element: that negative functional state of nervous elements of the region answers to the patient's loss of some speech. His utterances, positive element, however inferior as speech, answer to activities of nervous arrangement of Broca's region which are healthy—which are in all ways normal except possibly for slight "loss of control." Here is another illustration of the statement that part of the symptomatology of nervous maladies is the outcome of activity of healthy nervous arrangements.[67]

We must bear well in mind that speech is a psychical process. Nowhere is it more important to distinguish the psychical from the physical. I say once more that psychical symptoms are to medical men only signs of what is wrong in a material system. Our task as physicians is to ascertain the nature of the physical process correlative with speech, or more exactly the nature of the

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[66] My present concern with aphasia is only a very limited one. I need not always supply the obvious qualifications to statements made in the text. The expressions "loss of some speech" and "retention of the rest of speech" must not be taken literally as if they meant that the patient had lost certain words or propositions of the (his) English language altogether, and had retained the rest of them intact. Such is an impossible condition of things when an aphasic patient corrects his mistakes. But the statements may be taken literally for the mere purpose of a limited illustration. I do not suppose that there are fixed nervous arrangements– some for these words or syllables (properly movements corresponding to syllables) only and some for those only. I would rather than hold this mechanical doctrine go to the other extreme, and say that there are no nervous arrangements for movements in any centres except at the time when these and those motor nervous elements are functioning together in a particular temporary grouping.

[67] It is one thing to locate the negative lesion which destroys speech (renders a person unable to speak aloud), and quite another thing to say that "speech resides" in any particular part of the cortex. Words, or some other symbols, serve us during thought; when a man is thinking "gold is yellow," words in propositions are as certainly concerned as they are when he says that aloud. I submit that the highest centres ("organ of mind") must be engaged during speech, whether external or internal, notwithstanding that a negative lesion of a part of the middle motor level produces aphasia. Not believing in abrupt localisations, I do not mean by using the term "Broca's region" to limit the negative lesion productive of aphasia to that part of the lowest frontal convolution which enters into the "motor region." As is the custom, I shall neglect

concern which the highest centres must have with speech.

anatomical substrata of words (syllables). No one denies that the physical basis is of cells and fibres, but this is a morphological account of it. The anatomical basis is, I submit, of morphological elements so grouped as to form certain sensori-motor nervous arrangements. These, I presume, are audito-articulatory—that is, they are nervous arrangements representing certain auditory impressions along with certain corresponding complex (articulatory) movements of the tongue, palate, lips, etc. We suppose the *motor* elements of these *sensori-motor* arrangements to be of Broca's region. In the case of Defect of Speech E our ultimate concern is with the two opposite functional states of these motor elements which answer respectively to the two opposite psychical elements of the double symptomatic condition, which is untortunately named (defect of speech or partial aphasia) after the negative element only. The motor elements alone of the sensori-motor substrata of speech are damaged. I submit that in Defect of Speech E there is *correlative with* its negative psychical element exhaustion of some nervous arrangements of Broca's region, which is productive of loss of some of the complex movements of the muscles of the tongue, etc. ("articulatory muscles"). And I submit that the speech remaining possible is correlative with integrity of other nervous arrangements of that same region for other complex movements of the same muscles; these healthy nervous arrangements function during the inferior speech remaining possible to the patient, and produce the corresponding articulatory movements. I have now to support the conclusion that the inferred exhaustion of some elements of Broca's region in Defect of Speech E produces paralysis in the sense of loss of complex movements of the tongue, etc.

There is often with Defect of Speech E some temporary decided paralysis of (loss of many movements of) the right arm. This statement, however, although allowable clinically, is not a scientific one. We should not in a realistic inquiry group together a psychical symptom and a physical one as if when so considered they were symptoms of the same order. In harmony with what I have said, the things really comparable are respectively loss of some complex movements of the muscles of the tongue, etc. (corresponding to the negative element in the defect of speech), and loss of some complex movements of the muscles of the arm (in both of movements represented by the middle motor centres). In this sense the two co-existing things are paralyses. On this basis alone is their concurrence intelligible and their comparison and contrast possible. With regard to the defect of speech, it is to be vividly kept in mind that the fellow part of the right middle motor centres (centres of the left half of the brain) for other complex movements of the muscles of the tongue, etc., and that the right and left lowest (bulbar) motor centres for simplest movements of those muscles are intact.

Those who do not follow me in distinguishing the psychical from the physical will, perhaps, think that I am confounding difficulty of articulation, a physical symptom, with a mental symptom (with "aphasia"). The fact is that I am making an absolute distinction between speech, which is a psychical process, and its correlative anatomico-physiological process, which is purely physical. It is only the *physical bases* of words (or properly of syllables) which I assert to be sensori-motor nervous arrangements representing complex movements of the muscles of the tongue, etc., in association with complex combinations of auditory impressions. I only say that *correlative with* the negative psychical element in defect of speech there is the *physical loss* of some complex move-

ments of the tongue, etc. Still, it may be thought that I have no clear ideas on the difference between aphasia, a mental defect, and, for example, "bulbar paralysis." In reality I have said that the "bulbar centres" (some lowest motor centres) are quite intact in Defect of Speech E. In bulbar paralysis there is loss of the simplest movements of the tongue, etc., and the negative lesion is bilateral (of certain motor centres of both halves of the lowest level), whereas in all cases of aphasia there is loss of complex movements of the tongue, etc., and the negative lesion is of motor centres of but one (left) half of the middle level, and the lowest motor (bulbar) centres are intact. Is not this difference enough?

The objection may be made that, whilst in the case of post-epileptiform paralysis with partial aphasia there is obvious paralysis of the arm, there is, as a matter of fact, none of the tongue, palate, lips, etc. It will be well in further exposition to stop speaking of Defect of Speech E, and to take in illustration a simpler (although much more serious) case, one of *loss* of speech ("complete aphasia") from a destructive lesion, say from softening of Broca's region. I shall suppose, however, that there is retention of "yes" and "no."[68] Here the negative element is loss of nearly all speech; the positive element being only retention of "yes" and "no." I shall call this case Loss of Speech D, or Complete Aphasia D; but the word "loss" is to be taken as qualified by "except for retention of 'yes' and 'no.'" We shall suppose there to be, as there nearly always is, right hemiplegia.

To say that the patient Aphasia D is speechless because he has lost the memory of words is to give no explanation of his inability to speak, any more than it is an explanation of the paralysis of his right arm and leg to say that it is owing to loss of volition. All psychological explanations of physical inabilities are merely verbal. Admitting for the sake of argument only that loss of memory of words is a correct *description*, our concern as physicians is with the physical condition correlative with that psychical loss. A part of the patient's brain has disappeared, probably the hinder part of the lowermost (left) frontal convolution (and no doubt more brain adjacent) has been changed to defluent stuff which is brain no longer; we shall speak of this as "destruction of Broca's region." I repeat that the lesion causes paralysis. I admit that there is no disability in the muscles of the tongue, palate, lips, etc., in their commonplace services, as in eating, drinking, swallowing, etc.; I admit it readily, because I only mean that there is loss of the complex articulatory movements of those muscles. There is nothing discoverably wrong with these muscles except for the slight facial and lingual paralysis which is a part of hemiplegia, right or left. The case illustrates that we may have loss of some *movements* of certain *muscles* without discoverable disability of those muscles. The seeming paradox in the particular case under remark is easily explained by Broadbent's well-known hypothesis.

In our speechless patient, not only are the lowest most motor centres of *both* halves of the bulbar part of the lowest level for simplest movements of the muscles of the tongue, etc., intact, but

[68] To speak is not merely to utter words, but *to propositionize*. The patient spoken of in the text, although not absolutely speechless, is so, except that he has only the use of the two most general of all propositions; I say "the use of" supposing that in the case taken for illustration the patient can assent and dissent by them. If he only uttered them at random, or if they were only signs of emotion, they would not serve propositionally, they would then be of no speech value.

also, what is most important, complex movements of these muscles of both sides remain represented in the right half of the brain (middle motor centres). Hence, on account of those remaining representations, there is no obvious disability of the muscles of the tongue, etc., in their commonplace services. Taking the simple representation by the lowest level for granted, the assumption is that the muscles of both sides of the tongue, palate, lips, etc., are represented by complex movements in each half of the brain, though no doubt more especially in Broca's region than in the fellow-region of the right. Here is an exemplication of Broadbent's hypothesis. Horsley and Schäfer write: "The *face area*, although we have so called it for convenience sake, actually gives rise to movements not only of the facial muscles, but also of the whole of the upper part of the alimentary tube (mouth, throat, and larynx)..... It is physiologically remarkable from the fact that many of the movements which result from its excitation are apt to be executed bilaterally, which is only exceptionally the case with excitation of the other areas (except that for the head and eyes)."[69] Hence destruction of neither Broca's region nor its fellow-part of the right middle motor centres produces disability in the commonplace services of the muscles of the tongue, etc., although beyond all question some movements of them are lost. But destruction of part of both halves does,[70] as some cases of double hemiplegia show, notably one recorded by Dr. Thomas Barlow.[71]

So that, so far from "confounding cases of aphasia with cases of bulbar paralysis," I am making a very definite distinction. So far am I from comparing loss of speech, considered as a negative psychical symptom, with the physical symptoms of bulbar paralysis, that I urge that there is not even a basis for any reasonable contrast of the two; psychical symptoms and physical symptoms are "not on the same platform." Regarding the physical condition correlative with the aphasia and that of the case of bulbar paralysis, the cases are alike in that there is in each loss of movements of the same muscles; on this basis I urge their resemblance as cases of paralysis. At the very same time I urge the differences between loss of complex movements of the tongue, etc., represented by certain *middle* centres of but *one* half (left) of the brain in cases of Loss of Speech D, and loss of simplest movements of those parts represented by certain *lowest* (bulbar) centres of *both* halves of the lowest level in cases of bulbar paralysis.

Furthermore, the tongue, palate, and lips not only act well in eating, drinking, swallowing, etc., in Loss of Speech D, but also when the patient says "Yes" and "No." He may have a stock utterance, as "Awful," "Oh, my God!" etc. He may when excited swear or get out other elaborate ejaculations; none of these are speech proper—they are not propositions, but compound interjections; the patient cannot repeat ("say") what he ejaculates. The articulation of all these utterances is perfect, and I presume that no one would deny that the articulatory movements are effected by perfectly healthy nervous arrangements. They are effected, I submit, by those of the undamaged right half of the brain, of course with subagency of lowest (bulbar) motor centres of both halves. Still neglecting, as everybody else does, the concern of the highest centres with speech and with morbid affections of it in disease, I submit that as regards speech the right

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[69] *Phil. Trans.*, 1888.

[70] Such cases are sometimes called cases of cortical bulbar paralysis; they are double cortical; the bulbar centres are intact.

[71] JOURNAL, 1877.

half of the brain is the automatic half, and that the left is that half in which automatic action ceases into what we call voluntary action.

The positive condition of our patient, Loss of Speech D, is that he retains the propositional use of "yes" and "no," physically two complex articulatory movements of the tongue, etc.

Now I draw attention to the fact that the patient who never speaks, never does make (with the exceptions stated) the complex movements of articulation, which I say are lost. Instead of explaining that he does not do so *because* he has lost "the memory of words," I submit that he never makes them because the nervous arrangements for those movements have disappeared—that he has not got those complex movements. Once more I urge that what is psychically aphasia is physically paralysis.

Moreover, the part destroyed in the case of aphasia is motor; no one denies since the researches of Hitzig and Ferrier that the homologous part in lower animals is motor. Hence, there is nothing for destruction of it to produce in these animals except paralysis. Mark that although destruction of that part of the "motor region" on the left in man produces no disability of the muscles of the tongue, etc., of either side (beyond the slight right facial and lingual paralysis spoken of as part of hemiplegia), discharge of that part develops movements of these muscles of *both* sides; by subagency, of course, of certain lowest motor centres of both halves of the lowest level, develops a contention of complex and of simplest movements of those muscles. From discharge of the part on the left are revealed the movements, the loss of which, upon destruction of that part, is masked by compensation by the fellow part of the right half of the brain. Yet there may be discharge (impulses passing by *route* of the callosal fibres) of the fellow part on the right consequent on the primary discharge of the left. I mention again that Sherrington has traced degenerated fibres from a unilateral lesion of the lower end of the fissure of Rolando into both halves of the pons and medulla.

Hence I conclude that the patient hemiplegic and speechless (aphasic) has not only lost very many complex movements of the muscles of the right arm and leg, but that he has also lost many complex movements of muscles of both sides of the tongue, lips, etc.; the conclusion is that his whole physical condition is, and is nothing else than, paralysis *in the sense of loss of movements*.

The condition is fundamentally the same in the Defect of Speech E, but the negative element (loss of some speech) is less, and the positive element (retention of much speech) is greater. Here, again, it will be well to illustrate by the case of a destructive lesion (a much smaller one than in Loss of Speech D), by softening, destroying but little of Broca's region; I will call this case Defect of Speech D. There is not rarely from such a pathological process a condition essentially like that in some cases of Defect of Speech E. Let us suppose that in the case of Defect of Speech D the patient frequently makes mistakes when he talks, either by using wrong words or by being reduced to very "automatic" utterances, such as "Very well," or that he often uses roundabout expressions such as "It won't come out here" (which, eked out by appropriate pantomime, meant "I can't make water"). And yet, to simplify exposition, I will take the patient's saying "hat" for "carpet" only, that to be a sample of the whole of his condition. Here we see the necessity of distinguishing the two elements, negative and positive. It is certain that the softened brain (really what is brain no longer; there is a hole filled with detritus of nervous elements) cannot be the cause of the patient's saying "hat;" that

is the speech left, and is owing to activity of the part of Broca's region which remains perfectly healthy.[72] This, his positive condition in speech, is sampled by his saying "hat." His negative condition is alone owing to "disease," and is sampled by his inability to say "carpet."

The negative element in Defect of Speech D is a minor degree of that in Loss of Speech D; the positive element is a major degree of the positive element ("Yes" and "No" only) in Loss of Speech D; in the former the dissolution of Broca's region is shallow and the level of evolution high, whilst in the latter the dissolution is very deep and the level of evolution very low, if there be any in that region. The two cases differ in double degree. In the Defect of Speech D there is much speech left, and there are very many mistakes; in the Loss of Speech D there is very little speech left, and there are no mistakes, the patient correctly assenting to and dissenting from (by "Yes" and "No") all statements made to him.

Now for the physical condition in Defect of Speech D. Answering to the slight negative element, there is loss of but a few complex articulatory movements represented in Broca's region; answering to the positive element there is retention of many other such movements; or we may say, limiting illustration to the samples, there is loss of the complex movements answering to inability to say "carpet," and retention of the complex movement answering to saying "hat." If anyone says that a word (properly a syllable) is not a movement he is agreeing with me; for I am contending that activity of nervous arrangements representing certain complex movements (with, of course, corresponding impressions) of the tongue, etc., is only the anatomico-physiological process going on during the appearance of what are the psychical things—words. I need only say that the softening in Defect of Speech D is the negative lesion equivalent to the exhaustion in Defect of Speech E. The utterances both in Defect of Speech E and D are the outcome of activities of perfectly healthy nervous arrangements. The negative lesion in both is dissolution; in each the speech, however imperfect, as certainly signifies evolution going on on a lower level, as the speech of healthy persons does evolution going on on the normal level.

I have admitted that the patient in Defect of Speech E and D may correct his mistakes—that after saying the wrong word "hat" he may immediately say the right word "carpet,"—and I will here admit that when he says "hat" he knows that the word is not the right one. But this does not invalidate the principle of the explanation. For convenience of exposition I made the arbitrary assumption that there are fixed and unalterable nervous arrangements for particular movements, in this case for complex movements of the tongue, etc., corresponding to syllables. I think the seeming difficulties could be met if, abandoning that assumption, I were to go into detail as to the constitution of nervous centres. At any rate, the attempt is to give a materialistic explanation of the physical condition in aphasia, without confounding the concomitant psychical states with that physical condition. I submit that the explanation is, at any rate, as good as the psychological explanation. If we do invoke the latter so-called explanation, we have not only to account for, to take the samples again, "loss of memory" for "carpet," but also for the

[72] To give another example, surely it is impossible that when the patient referred to in the text said, "It won't come out here," there was engagement of nervous arrangements which were "diseased" in any way whatever; the "disease" was the cause of his inability to say "I can't make water."

"over-memory" for "hat." When the patient cannot say "carpet," why does he utter anything? Why does "hat" come out instead? And admitting that the "loss of memory" for "carpet" is only temporary, and that next moment the patient can say "hat," we have to account for the temporary defect of "memory" and for its sudden restoration for that case. Moreover, if the patient says "hat" and knows that it is wrong, he can only know it by there being revived in him the word "carpet" at the very same time; certainly these two words must be revived mentally; there is then "verbal diplopia." If it is admitted that the psychological statements as to the aphasic conditions are correct descriptions, those who make them are just as much bound as anyone else to seek the abnormal material conditions of the several phenomena of "amnesic aphasia." And bearing in mind the proof given by Ferrier, Horsley, Schäfer, Beevor, Sherrington, and many others, that the region damaged is motor, a merely morphological account of the physical condition in aphasia will not suffice.. If anyone says that he cannot understand how activities of motor nervous arrangements can correspond to words, I would remind him that, except the popular psychologist, no one pretends to understand how any material conditions correspond to any psychical states.